TRANSACTIONS

OF

The Association of

Life Insurance Medical Directors

of America

FIFTY-SEVENTH ANNUAL MEETING

James R. Gudger, M. D.
Editor

VOL. XXXII

PRESS OF
Recording & Statistical Corporation
New York City
1949

OFFICERS OF THE ASSOCIATION, 1947-1948

ALBERT J. ROBINSON, M. D. ..... *President*
EDWIN G. DEWIS, M. D. ..... *First Vice-President*
HARRY E. UNGERLEIDER, M. D. ..... *Second Vice-President*
HENRY B. KIRKLAND, M. D. ..... *Secretary*
WALTER A. REITER, M. D. ..... *Treasurer*
JAMES R. GUDGER, M. D. ..... *Editor of the Proceedings*

EXECUTIVE COUNCIL

A. J. ROBINSON, M. D.
E. G. DEWIS, M. D.
H. E. UNGERLEIDER, M. D.
H. B. KIRKLAND, M. D.
W. A. REITER, M. D.
J. R. GUDGER, M. D.
E. C. BONNETT, M. D.
B. F. BYRD, M. D.
L. H. LEE, M. D.
J. M. LIVINGSTON, M. D.
J. B. STEELE, M. D.
L. S. YLVISAKER, M. D.
R. L. ROWLEY, M. D.
H. M. FROST, M. D.
D. E. W. WENSTRAND, M. D.
WILLIAM BOLT, M. D.
S. J. STREIGHT, M. D.
A. O. JIMENIS, M. D.

OFFICERS OF THE ASSOCIATION SINCE ITS ORGANIZATION

Presidents

John M. Keating, M. D.	1889-1891
Frank Wells, M. D.	1891-1894
Edgar Holden, M. D.	1894-1897
H. Cabell Tabb, M. D.	1897-1899
George R. Shepherd, M. D.	1899-1902
Edward H. Hamill, M. D.	1902-1904
John W. Fisher, M. D.	1904-1906
Oscar H. Rogers, M. D.	1906-1908
Thomas H. Willard, M. D.	1908-1910
George Wilkins, M. D.	1910-1911
Brandreth Symonds, M. D.	1911-1912
Harry Toulmin, M. D.	1912-1913
Edward K. Root, M. D.	1913-1914
William E. Porter, M. D.	1914-1915
Franklin C. Wells, M. D.	1915-1916
Homer Gage, M. D.	1916-1917
Walter A. Jaquith, M. D.	1917-1918
Thomas H. Rockwell, M. D.	1918-1919
Faneuil S. Weisse, M. D.	1919-1920
Augustus S. Knight, M. D.	1920-1921
Thomas F. McMahon, M. D.	1921-1922
Frank L. Grosvenor, M. D.	1922-1923
William R. Ward, M. D.	1923-1924
Chester F. S. Whitney, M. D.	1924-1925
Angier B. Hobbs, M. D.	1925-1926
Wesley W. Beckett, M. D.	1926-1927
Robert M. Daley, M. D.	1927-1928
J. Allen Patton, M. D.	1928-1929
William Muhlberg, M. D.	1929-1930
Robert L. Rowley, M. D.	1930-1931
Charles L. Christiernin, M. D.	1931-1932
H. Crawford Scadding, M. D.	1932-1933
Robert A. Fraser, M. D.	1933-1934
Fred L. Wells, M. D.	1934-1935
Eugene F. Russell, M. D.	1935-1936
Chester T. Brown, M. D.	1936-1937
Samuel B. Scholz, M. D.	1937-1938
Henry W. Cook, M. D.	1938-1939
Harold M. Frost, M. D.	1939-1940
Donald B. Cragin, M. D.	1940-1941
David E. W. Wenstrand, M. D.	1941-1942
William Bolt, M. D.	1942-1945
Samuel J. Streight, M. D.	1945-1946
Albert O. Jimenis, M. D.	1946-1947
Albert J. Robinson, M. D.	1947-1948

OFFICERS OF THE ASSOCIATION

Vice-Presidents

Gurdon W. Russell, M. D.	1889-1892
Lewis McKnight, M. D.	1889-1892
John W. Fisher, M. D.	1892-1895, 1902-1904
H. Cabell Tabb, M. D.	1892-1897
Frederick W. Chapin, M. D.	1895-1896
Joseph H. Webb, M. D.	1896-1899
George R. Shepherd, M. D.	1897-1899
Edward H. Hamill, M. D.	1899-1902
Morris L. King, M. D.	1899-1902
Thomas H. Willard, M. D.	1902-1908
Oscar H. Rogers, M. D.	1904-1906
George Wilkins, M. D.	1906-1910
Robert L. Lounsberry, M. D.	1908-1909
George A. Van Wagenen, M. D.	1909-1910
Brandreth Symonds, M. D.	1910-1911
Harry Toulmin, M. D.	1910-1912
Edward K. Root, M. D.	1911-1913
William E. Porter, M. D.	1912-1914
Franklin C. Wells, M. D.	1913-1915
Homer Gage, M. D.	1914-1916
Walter A. Jaquith, M. D.	1915-1917
Arthur B. Wright, M. D.	1916-1918
Thomas H. Rockwell, M. D.	1917-1918
Faneuil S. Weisse, M. D.	1918-1919
Augustus S. Knight, M. D.	1918-1920
Thomas F. McMahon, M. D.	1919-1921
Frank L. Grosvenor, M. D.	1920-1922
William R. Ward, M. D.	1921-1923
Chester F. S. Whitney, M. D.	1922-1924
Angier B. Hobbs, M. D.	1923-1925
Wesley W. Beckett, M. D.	1924-1926
Robert M. Daley, M. D.	1925-1927
J. Allen Patton, M. D.	1926-1928
William Muhlberg, M. D.	1927-1929
Robert L. Rowley, M. D.	1928-1930
Charles L. Christiernin, M. D.	1929-1931
Lawrence G. Sykes, M. D.	1930-1932
H. Crawford Scadding, M. D.	1931-1932
Robert A. Fraser, M. D.	1932-1933
Fred L. Wells, M. D.	1932-1934
Eugene F. Russell, M. D.	1933-1935

OFFICERS OF THE ASSOCIATION

Vice-Presidents — Continued

Chester T. Brown, M. D.	1934-1936
Samuel B. Scholz, M. D.	1935-1937
Henry W. Cook, M. D.	1936-1938
Harold M. Frost, M. D.	1937-1939
Donald B. Cragin, M. D.	1938-1940
David E. W. Wenstrand, M. D.	1939-1941
William Bolt, M. D.	1940-1942
Samuel J. Streight, M. D.	1941-1945
Albert O. Jimenis, M. D.	1942-1945
Albert J. Robinson, M. D.	1945-1947
Edwin G. Dewis, M. D.	1946-
Harry E. Ungerleider, M. D.	1947-

Secretaries

Frank Wells, M. D.	1889-1891
Elias J. Marsh, M. D.	1891-1894
Oscar H. Rogers, M. D.	1894-1900
Brandreth Symonds, M. D.	1900-1907
William E. Porter, M. D.	1907-1912
Faneuil S. Weisse, M. D.	1912-1918
Angier B. Hobbs, M. D.	1918-1923
Chester T. Brown, M. D.	1923-1931
Edwin G. Dewis, M. D.	1931-1946
Henry B. Kirkland, M. D.	1946-

Treasurers

John W. Brannan, M. D.	1889-1899
Frank S. Grant, M. D.	1899-1903
Augustus S. Knight, M. D.	1903-1918
Charles L. Christiernin, M. D.	1918-1929
Albert O. Jimenis, M. D.	1929-1942
Walter A. Reiter, M. D.	1942-

Editors of the Proceedings

Angier B. Hobbs, M. D.	1917-1918
Robert M. Daley, M. D.	1918-1923
Eugene F. Russell, M. D.	1923-1928
Albert O. Jimenis, M. D.	1928-1929
Robert A. Fraser, M. D.	1929-1932
Samuel B. Scholz, M. D.	1932-1935
Harold M. Frost, M. D.	1935-1937
Harry E. Ungerleider, M. D.	1937-1947
James R. Gudger, M. D.	1947-

Transactions
of
The Association of
Life Insurance Medical Directors

FIFTY-SEVENTH ANNUAL MEETING

The Fifty-seventh Annual Meeting of The Association of Life Insurance Medical Directors of America was held at the Hotel Pennsylvania in New York City, on Thursday and Friday, October 28 and 29, 1948.

PRESIDENT ROBINSON — It would have been a great pleasure and privilege to have held this Fifty-seventh Annual Meeting of The Association of Life Insurance Medical Directors in my home city of Hartford. We could have provided a good auditorium, fine food, and some entertainment. However, the expansion of the insurance business and other industries in the Hartford area has temporarily outgrown adequate hotel facilities for a meeting as large as this one. We are not strangers to the Hotel Pennsylvania. On previous occasions, this Association has been well served. This year, I am sure, will be no exception to that rule.

The problems of medical selection cover every conceivable type of human impairment. It is difficult enough for most of us, with our multiple duties, to keep informed on the ever-changing conditions which affect mortality and morbidity.

It is not enough that we study conditions as they affected mortality twenty years ago, or even as they affect mortality today. We must endeavor to discover, if possible, the trend of events and conditions as they may influence the future.

Our annual meetings, therefore, afford an important answer to this problem of keeping the medical director alert to changing conditions.

We meet for the purpose of exchanging ideas, without which little human progress is possible. Each year brings new problems to be solved. This year is no exception.

In formulating the program for this meeting, it has been my aim to present to you authoritative and practical information relating to our overall selection problem. Our guest speakers are outstanding in their respective fields. We deeply appreciate the time, effort, and thought these men have put into this program. All of the material they present to us will be beneficial in carrying out our responsibilities to our respective companies. We shall hear from a chief executive, who must keep his eye on all factors which affect favorably or unfavorably the successful operation of a life insurance company. We shall hear from an actuary who is an expert on statistical analysis. We shall hear from members of our own Association who are thoroughly competent in the field of life insurance medicine. Most of us have been or will shortly be faced with problems arising out of the atomic age. I, for one, admit to a sketchy knowledge of this field. The scientist, the doctor, and the statesman will present material to help us formulate our thinking on how atomic energy presently affects our job of risk selection and how it may affect it in the future.

We have two full days of scientific program ahead of us. I shall deeply appreciate the cooperation of the membership in being on time for the meetings, because we intend to start on schedule. Thank you.

This program will be opened by a man with whom I have had the pleasure to be associated for twenty-five years. He has devoted his entire business life to insurance in general and to the Connecticut General in particular. Mr. Wilde has been identified with the Institute of Life Insurance from its inception. He was chairman of the Primary Survey Committee, chairman of the Organization Committee, and first chairman of the Board of Managers. He is a member of the Executive Committee of the American Life Convention and the Insurance Committee of the United States Chamber of Commerce. He is a director of several corporations, and has also

found time to serve his home community as chairman of its Board of Finance. It seemed to me that it would be refreshing and instructive to have a chief executive tell us where medical directors and their associates fit into the broad pattern of life insurance. It will be interesting to see if we see ourselves as others see us. It is my pleasure to introduce Mr. Frazar B. Wilde.

A LAYMAN LOOKS AT THE MEDICAL DEPARTMENT

FRAZAR B. WILDE
President
Connecticut General Life Insurance Company

One of the conventions of our day is for the speaker to register his pleasure in his audience. In this instance that can be done with sincerity for a number of reasons. Not the least of those reasons is the opportunity which all of us cherish to explain to the other fellow how he ought to run his business! Dr. Robinson was kind enough to give me that opportunity and I propose to take full advantage of it! Because talks have titles, this one may be described as "A Layman Looks at the Medical Department."

Now it would be a fair question from the medical directors' point of view to ask why a layman looks at the Medical Department. A layman should have some hesitancy in approaching a professional area. However, the chief executive of a life insurance company must look at the Medical Department because it is a significant sector of his responsibility.

The operation of life insurance is different from the majority of businesses. Many businesses have two major components —one is production, the other is sales. The life insurance business has three major elements. They are all vital in the successful conduct of the business. One is sales, another is underwriting, the third is investment.

The chief executive may be less familiar with the technicalities of modern insurance underwriting than he is with investment or sales. He does need to understand something about the principles and the policies involved, and he certainly must be able to assure himself that those directly charged with underwriting responsibility are competent to perform. None of us whose service in this business represents any appreciable

length of time can fail to recall that there have always been companies whose directors and executives learned this lesson the hard way.

Before an executive can determine the abilities required to carry out a certain function, it is obvious that the nature and quality of the operation must be established. What is the function of the modern Medical Department? You gentlemen well know that the Medical Department's chief object is not to make a field man unhappy by declining a risk.

While it is true that the principal function of the Medical Department is underwriting of risks, the nature and complexity of that job is infinitely greater than the normal concept of it. Perhaps a good definition would be to say that a modern Medical Department tries at all times, in cooperation with developments on the lay side, to issue insurance in appropriate amounts and at a proper price to the highest possible number of applicants. Figures are hard to establish on a comparable basis. It is safe to say that if all the prospects for life insurance whose applications were submitted through a well-trained agency force furnished all of the information requested and took whatever additional tests might be required, insurance on some plan and at some price would be offered to more than 95 per cent of those seeking it.

As a matter of sound field relations and as a matter of good general public relations, it would be helpful to have it better understood than it now is that the life insurance business is ready, willing, and able to insure a surprisingly high percentage of the population. There are still too many agents and too many of the public who labor under the delusion that coldly, impersonally and with much scientific paraphernalia, the local examiner and the doctors at the Home Office combine in a conspiracy calculated to find many and frequent reasons for declinations. More can and should be done to emphasize the success that you gentlemen have made in developing and broadening underwriting. Such education is in the interest of good repute for the companies and the Medical Departments who have largely brought about this improvement. We have

an instrument in the life insurance business, namely, the Institute of Life Insurance, which could help if this suggestion has merit.

Now the fact that a constantly increasing percentage of people are able to buy insurance for some amount and at some price has added greatly to your responsibility for the continuous development of technical skill in medical underwriting, and it has added to a collateral problem; namely, it requires of the modern Medical Department the business courage to properly price the insurance offered. One of the embarrassments which field men do mention, and perhaps on occasion with some justice, is the wide spread which sometimes occurs in pricing insurance. That is particularly true if the risk has impairments which run in excess of 150 per cent rating. We are all sincere believers in individual competition. We expect price differentials and since the field of substandard, with high ratings, is relatively new, it is logical to expect some important differences. It is not reasonable when the difference in price arises because some agent was a better salesman than another in dealing with his Home Office. A modern Medical Department pretty much eliminates the personal sales talk revolving around the high standing of the applicant in his community, etc., as being an element entitled to any weight in formulating an underwriting judgment. In the long run, field men are happier and the Home Office has a great deal more self-respect because it is doing a professional job based on facts and on professional opinion and not salesmanship. That is not to say, from the point of view of the chief executive, that the Medical Department should be austere and not willing to listen. There is a vital necessity of affording a field man the opportunity to understand the facts in a case, and to understand that, unless by re-examination or some other review the facts are changed, you must stick to your professional judgment. There is quite a difference between the approach of assuming that the agent is either too young to know or too ignorant. That is something which no modern Medical Department will permit to creep into its relations with the field force, who are a vital and constantly improving element in the great business of life insurance.

If the principal function of the Medical Department is to select risks and price them properly, then it follows that the maximum practical use must be made of all the machinery that modern scientific medicine can offer. There are some limitations in this workaday world. On the other hand, many of these limitations can be overcome or mitigated. You will recall that when such requirements as extra heart studies or blood sugar tolerance tests were first instituted, it was clear to the Agency Department that such devices and extra requirements were certainly inventions of the devil calculated to harass the public and lose commissions for the agent! By internal salesmanship at the Head Office directed through the Agency Department, it is entirely practical to demonstrate that extra tests are constructive and are in the public's interest and in the agents' interest. When the Home Office Agency Department has that concept, it has the ability to sell it to the field. Agents with that leadership will sell it to the applicant. The ability of a good field man to carry out any Home Office policy is almost without limit provided he has been fully informed of the fairness and justification of such policy and if he knows that additional requirements are in the interest of issuing more insurance at a fair price and not designed simply for the entertainment of the Head Office Medical Department.

One of the very important duties of the modern Medical Department is to develop a competent staff of field examiners. This is not an easy task because of the area to be covered both in a geographical and in a numerical sense. Written manuals and other well prepared information are used by many companies and are undoubtedly a help. It is my impression that more could be done in the way of visitation. There is probably not sufficient personal contact between members of a Home Office staff and the group of examiners in the field. While visits to the various territories have obvious limitations, there is nothing to equal personal contact in building mutual understanding. It is not good business either from the standpoint of underwriting or from the standpoint of its impact on the public to have hasty, superficial examinations because the local doctor has not been properly sold on his responsibility.

Out of that arises, possibly, some of the financial friction which has come to my attention. Many things in life are smoothed away by personal contact, which on their face appear to represent money or problems that could not be solved except by more money. It is possible that we have not met our full responsibility to local examiners on the money side. However, it is my impression that with the growth of the insurance business, the amount of additional and steady work that good examiners obtain, we have not been too niggardly on an overall basis.

Today's Medical Department on the modern basis does not stop with underwriting risks and the determination of the proper price for the insurance. There is the matter of the Home Office and the Field Staff both in its initial employment and subsequent health check-up. There is the long-range problem of contributing to the general welfare of the country as a whole by participation in appropriate areas of medical research and development. We in the life insurance business probably should have close to a model operation in those areas. Whether or not we have may be open to further examination.

Probably the majority of companies today do examine new applicants for employment. Many have annual examinations. There are surprising gaps in this technique. Some companies do not examine field men; many do not examine for promotion. In the area of treatment of employees, we deal with a touchy problem. Most companies stop with preliminary techniques and simple treatment to relieve immediate symptoms. That seems to be the consensus of the minimum requirement. Whether a modern Medical Department should go further is somewhat a matter of individual company judgment or determination rather than a clearly established pattern. Whether some facilities such as x-ray or other forms of examination should be accorded families of the staff is another point for consideration on an individual basis. It has been my observation that with the constantly increasing demand for medical service, there is not very much criticism in local communities for an increase in the amount of service which a

given organization furnishes for its own staff. As a matter of fact, there are many large employers not in the life insurance business who are giving greatly increased facilities to their staffs, and more than most life insurance companies are doing today. That may not be quite in balance because we have some responsibility, I think, to do a model job, however that may be defined at any given time.

When we come to the general question of the promotion of medical science through a modern Medical Department, it seems to many that the best device for most companies is generous support of the Medical Fund. There is more than one instance where an individual company has participated locally both in money and in moral support of special projects. Certainly it is a benefit to the technical advancement of the Medical Department to have all of its members participate within reason in local medical activities, even though they do not normally and probably should not participate in any general practice regardless of the fact the hours in an office might sometimes suggest the opportunity to service the public in this way.

There is a matter touching on the Medical Department which needs emphasis despite the fact that it is obvious. With more and more being required of the Medical Department, it is more and more obvious that an adequate staff, both in numbers and in personal qualifications and in continuous training, is required. That is a subject which touches the chief executive as much as it does the Medical Department. The chief executive may not in every case have been fully informed of the needs of the situation.

There is a matter, last but not least in these remarks, which has become somewhat of an obsession with me, and that is the problem of measuring mortality in terms which are perfectly clear and intelligible to us, so that we may know where we are going. In the conduct of any business, it is interesting to know whence you have come, from a historical viewpoint. But, you cannot do a sound job unless you know what the current trend is.

We have used in the business, for many years, a mortality index which we call the "American Experience". We realized some time ago that it was confusing to the public, because we were able to talk about 40 or 45 per cent mortality, and that had the immediate suggestion that we had a terrific profit item in the business. Of course, you all know that is not so. Therefore, publicly we have pretty much curtailed the confusion by not using that figure.

But, curiously, within the business, in reports to our own boards of directors, discussions within small industry groups, we talk about the wonderful mortality measured in terms of the "American Experience". I know that in the case of our own company, in the first few months of the year we had something like 29 or 31 per cent. We all knew it was wrong and was not to be taken literally. But from my point of view, no matter how much we think we are rational humans we are subject to simple psychological tricks, and we cannot deal with 40 per cent mortality without thinking we are doing an outstanding underwriting job. And maybe we are.

We are perfectly sure that through the brilliant advances of medical science we have made progress each decade, particularly in the younger ages. But we know from some studies made, if we re-examine mortality on a realistic table, we will find that instead of 40 per cent it is of the order of 80 to 90 per cent, because the old table is simply taking the company's experience almost from the beginning of time. It represents all the business on the books.

What you gentlemen want to know and what any chief executive wants to know is the current mortality. This should cover the most recent figures that have significance and certainly should not be more than ten years old in all probability, or even five years.

Now, it is not my impression that our underwriting is reckless. It is my impression that the general improvement in health and the use of some of these figures have given us a slightly false sense of security. I do not believe that mortality experience is necessarily quite as good as we think it

is, and this has involved some optimism in underwriting, I am perfectly sure. I am doubly sure of that because of my own experience.

I am an underwriter in the security and mortgage field and we have had very few losses for ten to fifteen years. I am perfectly sure that some of our underwriting is not wise and I feel equally sure that this same element of deception can and will affect us in life insurance mortality unless we get some better indices to the true mortality trend.

In closing, may I say that the modern Medical Department is surely the foundation and the largest factor in life insurance underwriting. No matter what we may do in helping overhead by arithmetical devices, in small cases, we have got to depend for the conduct of this business on the highly trained professional skill, and the administrative distribution of our skill through our staff for good results. You have a challenging, a vital, and an important future.

PRESIDENT ROBINSON—I think, Mr. Wilde, that this audience has demonstrated its appreciation in the applause which your fine talk received.

Dr. Tinsley R. Harrison received his A.B. degree from the University of Michigan in 1919, his M.D. degree from Johns Hopkins in 1922, and an honorary degree of Master of Science from the University of Michigan in 1940. His career, both as a clinician and as a teacher, has been distinguished. He has been associated with several outstanding teaching hospitals, including Peter Bent Brigham, Johns Hopkins, and Vanderbilt University. He is currently Professor of Internal Medicine and Chairman of the Department at Southwestern Medical College, as well as President of the American Heart Association.

Dr. Harrison will talk to us on "Degenerative Diseases: a Backward and a Forward Look".

DEGENERATIVE DISEASES: A BACKWARD AND A FORWARD LOOK

TINSLEY R. HARRISON, M. D.
Professor of Internal Medicine
Southwestern Medical College, Dallas, Texas

In this brief review of some of the aspects of degenerative diseases and processes, several questions will be discussed. First, what are the degenerative diseases? Second, what progress has been made? Third, what are some of the problems that seem to need solution? Mention will also be made of some of the progress which is likely to be made in the future. And, finally, there will be discussed a few of the practical problems which are faced daily by the medical insurance examiner.

What is the scope of the degenerative diseases? A definition might be something like the following: the degenerative diseases are those disorders of unknown causation seen commonly in elderly subjects. That is about as far as one can go. If we accept such a broad definition, it is clear that a great many diseases, including most of the things that kill people, fall into this group.

Among these, disorders of the cardiovascular system assume first rank, and include such conditions as hypertension, coronary arteriosclerosis, and senile heart failure (which may or may not be related to coronary arteriosclerosis). In addition, we have also disorders of the central nervous system, including cortical atrophy, senile dementia, and cerebral arteriosclerosis. It is not at all certain that senile dementia is always related to senile arteriosclerosis, although this is the current concept. Finally, there is Parkinson's disease. These are the most common of the degenerative diseases of the central nervous system, according to this definition.

In addition, there are cancer, diabetes and pernicious anemia, and one could go on mentioning rarer disorders.

When speaking of the progress that has been made, we think immediately of two of the dramatic advances made in

medicine in the past three decades, namely, discovery of the causes and treatment of diabetes and pernicious anemia. In thinking about the long-range attack on the degenerative diseases, it may be helpful to stop for a moment and ask why progress was made, and what clues bearing on methods of attacking the other degenerative diseases can be obtained from the advances made in pernicious anemia and diabetes.

First, it is of great interest that these discoveries were made in institutions specifically devoted to research. Heretofore, progress in clinical medicine had come largely from the efforts of the individual physician unaided by anything except his cerebral cortex, and it is still true that progress can be made by this means. But, by and large, the great progress of the present century has come from institutions which were designed to make that progess.

Again using pernicious anemia and diabetes as examples, the next point to be brought out is that advances have come from studies first on animals and then on man. Whipple studied the response of anemic dogs to diet, and then Minot and Murphy studied patients. Banting and his group started with dogs and then worked with patients. Therefore, if the past can be a clue to the future, one would think it likely that progress in other degenerative diseases will come in the same way—by a combination of animal and human research.

The brilliant advances in diabetes and pernicious anemia were made as the result of the united efforts of basic scientists and clinicians. In both instances, it was found that these diseases were due to internal deficiencies, either deficiency of insulin or of the hematopoietic principle. It is much easier to think of people, as they become older, developing a lack of something rather than an excess of something.

Patrick Henry said, "There is but one lamp by which my feet are guided, and that is the lamp of experience." If we can apply that principle to the likely developments in other degenerative diseases, we may expect progress to come in institutions specifically set up and designed for research. By teamwork on the part of basic scientists and clinicians working

with animals and patients, it is probable that the causes of many of the degenerative diseases will be found to fall in the category of deficiencies. These will probably not be dietary or external deficiencies. They are more likely to be biochemical, internal deficiencies.

Turning from diabetes and pernicious anemia to cancer, there have been some encouraging developments in this field. Of course, we are all interested in the progress that is being made in terms of public and governmental support of cancer research. In the past few years, for the first time, there seems to have been a favorable turn in the war against cancer. For the first time, we seem to be advancing rather than retreating. Statistics seem to show that cancer of the uterus and cancer of the breast are causing somewhat fewer deaths than before. If so, it would appear that as the result of the intensive educational efforts directed toward the public these diseases are being detected a little earlier.

With regard to the other types of cancer, the curve of mortality has not yet begun to turn downward. In all probability, the cancers which do not produce manifestations early (and that means most of the internal cancers, for example, those of the gastro-intestinal tract) will resist any progress until some more fundamental knowledge about cancer is available.

In regard to the degenerative disorders of the central nervous system, little progress has been made. There is an increasing body of evidence which, although not yet conclusive, indicates that our former tendency to ascribe cortical atrophy and senile dementia to cerebral arteriosclerosis was mistaken. Dr. William Dock has insisted (and I believe correctly) that a person may age in the cortical cells, the myocardial cells, the pancreas, and the bone marrow, as well as in the arteries.

Asymmetric focal disorders of the nervous system are usually due to vascular disease. Proof is lacking that the condition characterized by gradual decay in the intellect, resulting eventually in "senile dementia" is the result of arteriosclerosis of the brain. It may not be the blood vessels, but the brain cells themselves, that wear out and cease to function with age.

In any case, it is likely that we shall learn more about it in the near future.

As for arteriosclerosis in general, some useful knowledge is being developed. Apparently sclerosis of the arterioles is, in the main, a result of hypertension. However, it does occur in milder degrees with advancing age in the absence of hypertension. As regards sclerosis of the larger vessels, the rôle of heredity has been stressed for years. However, since members of the same family tend to eat the same food, we cannot be sure that heredity alone is responsible for the condition in certain families. Past experience would support the idea that members of a given family tend to wear out in a given spot, but as yet our knowledge of the rôle of heredity in relation to arteriosclerosis is meager.

Hypertension, which is so important in small vessel sclerosis, also affects large vessels, but to a lesser degree. The available evidence seems to indicate that hypertension causes acceleration of the process and is an aggravating cause of large vessel sclerosis rather than an underlying cause.

The most promising development in the field of arteriosclerosis is the rather impressive body of evidence accumulated in recent years on the possible rôle of diet. Whether the important factor is the intake of cholesterol *per se* or the total caloric intake cannot be stated with certainty at present. Space does not permit us to discuss this problem, but there seems to be more than a slight chance for important progress in the near future.

Let us consider disease of the heart itself. Some rather important developments have occurred in the broad national picture. One of the most significant of these is the support given to the study of cardiovascular disease by the life insurance companies. Another one is the change in policy of the American Heart Association from a purely scientific body to a voluntary health agency, with activities in terms of education of the public, fund raising, support of research, and local heart programs. The development of the National Heart Institute within the Public Health Service is certain to lead to acceleration of progress.

The statement is commonly made that disease of the heart is incurable. On the contrary, certain types of heart disease are curable. Thus the disorders due to thyrotoxicosis and thiamine deficiency are often curable. A number of forms of congenital heart disease are now curable, thanks to our surgical colleagues. Certain types of heart disease due to pericarditis are alleviated by resection of the pericardium. The mortality of subacute bacterial endocarditis has changed from 99 plus per cent twelve years ago to 20 or 25 per cent now. That is due to the use of penicillin.

Certain types of heart disease are preventable. Syphilitic heart disease is largely being prevented by the treatment of early syphilis. The incidence of syphilitic heart disease among private patients is dropping rapidly, and we may expect the same thing to occur in all levels of the population.

There is considerable evidence that while rheumatic heart disease may not be entirely preventable, recurrences of rheumatic fever can be prevented in large measure. The available data would seem to indicate that prevention of streptococcal infections by prophylactic doses of sulfonamides in an individual who has had rheumatic fever reduces the frequency of recurrence very markedly.

Thus far the strides in the field of heart disease have been made in regard to those forms which are not primarily degenerative. However, considerable progress has been made in the management of congestive heart failure, a common sequel of the degenerative disorders. An important advance is the principle of rigid sodium restriction which is possibly the most important practical advance in the treatment of cardiac diseases during the last decade. Many patients who would formerly have died in a few months can be kept alive for a great many months, and even years, if they follow a diet sufficiently low in sodium. The mechanism of sodium retention in heart failure is a disputed matter and one in which many people, including ourselves, are very interested. Much work is being done on it at the moment.

With respect to arterial hypertension, two methods of treatment which represent some advance have been developed

in the past fifteen years. Sympathectomy seems to be of definite value in certain patients. Selection of suitable cases is difficult, and we cannot tell which ones are going to improve and which ones are not. On the other hand, there is no question that life and usefulness have been prolonged in some patients by sympathectomy. With the rapid developments going on in regard to new drugs which depress the activity of the sympathetic nervous system, it may not be long before surgical sympathectomy will be replaced by medical sympathectomy. Possibly in a decade or two the operation will be discontinued and drugs substituted for it. That still is not the ideal treatment of hypertension, but it is something. The other method which we think has been of some value in a limited number of cases is the drastic restriction of sodium in the diet. It is effective only in a minority of cases, and it is useless in most of the middle-aged and elderly hypertensives. But there are some patients, particularly in the younger age group, in whom very dramatic results appear from marked restriction of sodium.

The success of the rice diet is apparently due entirely to the low sodium content. In rats with hypertension, one can obtain a marked lowering of blood pressure by using a diet low in sodium and containing no rice. If one gives a rice diet equally low in sodium, one observes an equally good effect on the blood presure of the hypertensive rat. If one utilizes a rice diet containing the same amount of sodium as the normal diet, one gets no effect at all on the rat's blood pressure. Therefore it seems likely that, in the case of rats, the rice diet is a low sodium diet and the effect is due to lack of sodium. Possibly the same is true in man. Unfortunately, sodium restriction affects only the minority of hypertensive patients. Why it is effective in some and not in others, we do not yet know.

What is going to happen if most people live to be eighty-five or ninety? If the older people have the same degree of vitality they now have on the average in that age group, a diminishing percentage of the population, (the younger group), will be supporting an increasing percentage of the

population, (the older group). Whether they do it personally and directly or indirectly through taxation, it is still the same thing. It is questionable whether it is worth while to prolong life unless one can prolong vigor. But we have every reason to think that if we do prolong the life span, we will prolong the span of vigor also. As the curve gets longer and longer, we shall face more and more frequently the problem of what to do with a man over sixty-five. A possible solution, but one which many say is contrary to human nature, is to educate people to believe that it is socially respectable to climb down the ladder. Have them believe it is no disgrace for a man at sixty-five to give up the presidency of the bank and become a vice-president, and that it is no disgrace for a professor to stop being a professor at sixty-five and become an associate professor, and at seventy become an instructor, and so on.

Finally, a word may be said, from the point of view of one who knows little about insurance medicine, about some of the practical problems relating to cardiovascular disease that you frequently face.

With regard to the value of blood pressure measurements, of family histories, and of body weight, there can be no serious question. The great difficulty in insurance medicine, as seen by an outsider, is that patients do not tell how they feel. In general, symptoms of disease tend to come before signs. In order to find out the status of a patient's heart the physician needs to know his history.

What ways are open to the insurance companies to compensate for inadequate histories? We all realize that physical examinations are often negative in the most serious forms of heart disease, that the electrocardiogram is often negative in people with coronary disease and even in patients with congestive heart failure. We realize that there are a good many people in the age group of 50 and over who have abnormal electrocardiograms and have perfectly good hearts. This abnormality may represent an old scar from previous cardiac injury but is not of any importance for the future.

The x-ray is a better diagnostic method when it is abnormal but is often normal despite the pressure of serious cardiac disease. In the majority of patients the history is far more important than is the electrocardiogram or the x-ray.

One wonders whether more would not be learned if every person over forty had to run up a flight of stairs of fifty steps, and if the doctor had to run with him. (The reason the doctor should run too is that a normal control is desirable. If the doctor is not normal, running up stairs would bring it to his attention early.) How short of breath does this patient become? How much more short of breath does he become than a normal man of the same age, weight, height and exercise habits? That establishes the level of myocardial reserve. No laboratory test will show it.

It is conceivable that one person in twenty-five thousand might be harmed by the exercise. But that is not likely to happen because those who had evidence of heart disease would not be exercised. And if one in twenty-five thousand is harmed, heart disease will be detected at an earlier stage in a great many applicants out of the twenty-five thousand. The usual danger in regard to heart disease lies in not knowing whether or not the person has it. It is worth the tiny risk in order to find out whether or not heart disease is present and, if so, to change the habits of living.

As to other suggestions, there are only two. If a physician were passing on the question of whether a man is apt to die of heart disease at some reasonable future date, he would want to determine the blood cholesterol. It would be useful to know whether it was abnormally high or abnormally low. It is possible that those who die of coronary disease and other forms of arteriosclerosis are going to be considerably fewer among those having normal or low blood cholesterol as compared to those with high cholesterol values. Determination of blood cholesterol would seem to be a worthwhile procedure.

Finally, the results of a very impressive study have recently been published by Dr. Isaac Starr, in Philadelphia, in which

he made ballistocardiographic studies on a group of apparently normal individuals ten or fifteen years ago. Now he reviews the status of those people. He finds there were a good many who were apparently normal ten years ago, except for certain changes in the ballistocardiogram, but who are now dead. Also, the incidence of cardiovascular deaths in a group of people apparently normal ten years ago, with certain ballistocardiographic changes, is very much greater than the incidence of deaths in subjects who did not show those ballistocardiographic changes.

Of course, the electrokymograph gives some of the same type of information. This instrument comes closer to giving information about the mechanics of the heart than any instrument we have had up to now, and it is my opinion that after the next five or ten years have passed, and we know more about the value and limitations of this instrument, we shall also learn to depend upon it for help.

The difficulty has been that up to the present time the only instrument available for evaluating heart function was the doctor's cerebral cortex and his pencil, or the patient's story. The electrocardiogram measures the conduction but not the contraction. The ordinary x-ray film reveals size but nothing about contraction. Physical examination helps but it gives little information on the way the heart is contracting. But the special instruments being developed now (the ballistocardiograph and the electrokymograph) are going to furnish a much better direct index to cardiac contractility than anything we have had before.

In summary: First, we do not know what diseases properly fall in the degenerative group. Second, we do not understand the causes of the ones we now include in the group. Third, although brilliant progress has been made in regard to some of the less common of the degenerative diseases, such as diabetes and pernicious anemia, only limited progress has been made in the more common degenerative diseases such as cardiovascular disorders and cancer.

If we can be guided by past experience, it is highly likely that when more is known about these important degenerative diseases, we will find them to be related in some way to deficiencies within the body.

There is still much to be done. There is a challenge for the future, if we are ever to achieve the state described by Browning in his poem, "Rabbi Ben Ezra," in which he said:

"Grow old along with me.
The best is yet to be,
The last of life
For which the first was made."

PRESIDENT ROBINSON—Thank you, Dr. Harrison. Dr. Harrison's splendid talk will be discussed by Dr. Lauritz S. Ylvisaker, Vice-President and Medical Director of the Fidelity Mutual.

DR. LAURITZ S. YLVISAKER—We are fortunate at this meeting to have the subject of Degenerative Diseases discussed for us by such an authority as Dr. Tinsley Harrison. Degenerative diseases today cause more deaths among life insurance policyholders than all other causes combined. They furthermore give us most of the problems which we have at the present time in life insurance selection. It is timely, therefore, that we briefly review the current mortality picture in order that we may see these degenerative diseases in their proper perspective and note some of the problems which they give us in selection.

We have been impressed with the importance of degenerative diseases from the study of our own death claims and from our daily experiences in the selection of new policyholders. Our mortality experience is relatively small but it has been critically analyzed and confirmed by repeated similar results. The figures here presented are the latest which the Fidelity has experienced, representing deaths during the first six months of this year.

Our knowledge of the basic fundamentals of disease is now so well advanced that we can group most all causes of death in a few relatively simple categories. The classification pre-

sented is based on these known facts; some individuals are born with impairments not compatible with life, giving rise to deaths from CONGENITAL MALFORMATIONS which concern us mainly in Juvenile Insurance. Those of us who are fortunate enough to be born relatively normal are exposed throughout life to the hazards of ACCIDENTS and INFECTIONS: If we escape these hazards, we meet with the hazards of growing old—the degenerative diseases. More than 95 per cent of our deaths during the last three years have fallen in these four simple categories.

TABLE I

CAUSES OF DEATH

THE FIDELITY MUTUAL LIFE INSURANCE COMPANY

JANUARY TO JUNE, 1948

518 LIVES

	NO.	%
1. CONGENITAL MALFORMATIONS	1	.2
2. ACCIDENTS AND VIOLENT DEATHS	22	4.3
3. INFECTIONS (TABLE 2)	42	8.0
4. DEGENERATIVE DISEASES		
a. CANCER (TABLE 3)	80	15.4
b. ARTERIOSCLEROSIS (TABLE 4)	355	68.6
5. MISCELLANEOUS (TABLE 5)	18	3.5
TOTAL	518	100.0

We cannot here enter into a discussion of the first three groups except by way of comparison. Not many years ago tuberculosis was the leading cause of death in this country and pneumonia ranked second as a cause of mortality. During the first six months of this year, less than 8 per cent of our deaths were due to tuberculosis, pneumonia and all other diseases of bacterial or viral origin.

TABLE 2

CAUSES OF DEATH	AGE AT DEATH BY LIVES								
	Under 30	30-39	40-49	50-59	60-69	70-79	80 & Over	TOTAL	% of TOTAL
INFECTIONS (other than cardio-vascular)									
Pneumonia	1	1	1	2	5	6	3	19	
Tuberculosis (Lungs 5, Other 1)	2	1	1	1	1			6	
Nephritis (Acute & Chronic)			2	2	1	1		6	
Appendicitis			1					1	
Hepatitis				1		1	1	3	
Bacillary Dysentery	1							1	
Meningitis	1							1	
Encephalitis					1			1	
Syphilis					1			1	
TOTAL	5	2	5	6	9	8	4	39	7.5

As the result of wonderful advances in the prevention of disease, we can soon look forward to almost complete eradication of tuberculosis in this country. With similar progress in therapy, we shall undoubtedly see a continued decline in the number of deaths from pneumonia and all infectious diseases. This result is a real tribute to all the forces which have been at work for many years for the improvement of public health.

The public health scene has, therefore, already shifted and will continue to shift more and more to the upper age group where we are exposed to the hazards of age. In 1948, over one-half of Fidelity Mutual policyholders had passed age 40, as is shown by the estimated distribution by attained age of lives of persons insured in Fidelity on June 30, 1948.

Attained Age		%
0–9	6,461	5.7
10–19	4,187	3.7
20–29	14,254	12.6
30–39	24,143	21.3
40–49	28,497	25.2
50–59	20,676	18.3
60–69	10,297	9.1
70–79	3,894	3.4
80–89	691	.6
90 and over	33	.1
	113,133	100.0

If we classify CANCER and ARTERIOSCLEROSIS with the degenerative diseases, we find that almost 85 per cent of our deaths were in this group in the first six months of this year. Fifteen per cent were due to CANCER and 68 per cent to the various manifestations of ARTERIOSCLEROSIS.

We cannot here enter into a discussion either of CANCER or ARTERIOSCLEROSIS, except briefly as they may affect us in our daily work of selecting new policyholders for the company which we individually represent. Our figures on deaths from CANCER (TABLE 3) are small but are comparable with larger groups in their relative frequency and age distribution.

TABLE 3

CAUSES OF DEATH	AGE AT DEATH BY LIVES							
	Under 30	30-39	40-49	50-59	60-69	70-79	80 & Over	TOTAL	% of TOTAL
TUMORS									
a. **Malignant**									
Gastrointestinal Tract (Stomach 13, Colon 16)			5	5	13	10	2	35	
Lungs		1	1	2	4	1		9	
Breast			1	4				5	
Kidney					2			2	
Bladder				1	1	4	1	7	
Prostate			1	2	3	4		10	
Leukemia					1			1	
Other			1	3	5			9	
b. **Non-Malignant**									
Brain (Post-operative)			1					1	
Prostate (Post-operative)						1		1	
TOTAL		1	10	17	29	20	3	80	15.4

They show that CANCER is particularly prevalent in the 40–65 age groups from which we draw many new policyholders, often for large amounts of insurance.

You may all have had experiences similar to the following:

CASE 1

APPLICANT:	MALE	AGE 56
HISTORY:	Negative except "recent hemorrhoids-recovery" (Attending Physician)	
EXAMINATION:	Negative	
POLICY:	$40,000.	
ISSUED:	11 December'47	
DEATH:	23 June'48	
CAUSE OF DEATH:	CANCER OF RECTUM	

In his declarations to our examiner, this applicant informed us that he had recently had what he thought was hemorrhoids and his attending physician confirmed this diagnosis and stated that the patient had recovered. In our investigation of this claim after death, we learned that the symptoms originally thought due to hemorrhoids had recurred and eventually a diagnosis of cancer of the rectum was made. He undoubtedly had a fully developed but still unrecognized cancer when we insured him.

CASE 2

APPLICANT:	MALE	AGE 43
HISTORY AND EXAMINATION:	Negative	
POLICY:	$6,000.	
ISSUED:	11 June'47	
DEATH:	6 September'48	
CAUSE OF DEATH:	CANCER OF SIGMOID	

In our investigation of this claim after death, we noted that he was examined on June 5th and that the policy was issued on June 11th. On June 9th, the applicant went to his doctor complaining of abdominal disturbances which were eventually diagnosed as being due to cancer of the lower bowel. He also must have had a fully developed but unrecognized cancer when we insured him.

Not long ago we had an application from a fifty-seven year old male who reported a recent prostatectomy with apparent

complete recovery. We had the policy ready to issue when we received a telephone call from his physician to whom we had written advising that his patient had cancer and that he had not informed the patient of this diagnosis.

These and other experiences force our attention to the fact; (1) that we are exposed to the hazard of unrecognized cancer in our consideration of applicants particularly in the 40–65 year age group, (2) that a seemingly innocuous history may prove to be significant and, (3) that attending physician statements covering such histories are very necessary.

It is our experience also that a careful examiner can often contribute much to the early detection of cancer by his findings on an insurance examination. We have made it a practice to report suspicious findings to the attending physician, with the result that leads given in our insurance examination have occasionally led to the early diagnosis of an unsuspected cancer.

In our consideration of ARTERIOSCLEROSIS as one of the important degenerative diseases, it should be emphasized that this condition is today the leading cause of death and that it will in the future take an even greater toll of lives. It is nature's way of terminating life. It should be considered primarily a disease of the arteries which develops with age and which eventually leads to narrowing and final occlusion of one or more arteries gradually decreasing or suddenly shutting off the blood supply to the various organs or parts of the body: the heart when the coronaries are occluded, the brain when the cerebral vessels are affected and the kidneys when the renal vessels are involved. During the last three years, between 60 and 70 per cent of our deaths have been due to these three most prevalent manifestations of arteriosclerosis.

It is unfortunate that this condition has loosely been labelled "heart disease" and has popularly become known as such. If we are to have a clear concept of circulatory diseases as we now know them and if we are to have comparable inter-company statistics of any value, we shall have to use a classification of these diseases based on pathology as we understand it today.

TABLE 4

CAUSES OF DEATH	AGE AT DEATH BY LIVES								
	Under 30	30-39	40-49	50-59	60-69	70-79	80 & Over	TOTAL	% of TOTAL
DISEASES OF HEART AND BLOOD VESSELS									
a. Congenital Malformations	1							1	
b. Infections									
Rheumatic		2						2	
Luetic									
Other (Pericarditis)					1			1	
c. Arteriosclerosis**									
Coronary (Occlusion & Other)		3	20	59	74	62	33	251	
Cerebral (Cerebral Hemorrhage)		1	4	9	19	35	13	81	
Renal (Chronic Nephritis)		2	1		4	4	4	15	
Other				1		3	4	8	
d. Other									
TOTAL	1	8	25	69	98	104	54	359	69.3
**Arteriosclerosis with Hypertension									
Coronary		1		15	18	11	5	50	
Cerebral		1	3	6	10	18	4	42	
Renal		1	1		1	2	1	6	
Other						1		1	
TOTAL		3	4	21	29	32	10	99	
%		3.0	4.1	21.2	29.3	32.3	10.1		
**Arteriosclerosis without Hypertension									
Coronary		2	20	44	56	51	28	201	
Cerebral			1	3	9	17	9	39	
Renal		1			3	2	3	9	
Other				1		2	4	7	
TOTAL		3	21	48	68	72	44	256	
%		1.2	8.2	18.7	26.6	28.1	17.2		

TABLE 5

CAUSES OF DEATH		AGE AT DEATH BY LIVES								
		Under 30	30-39	40-49	50-59	60-69	70-79	80 & Over	TOTAL	% of TOTAL
MISCELLANEOUS following International List of Causes of Death, Numbers I to XVIII.										
IV.	Anemia (Aplastic)		1						1	
V.	Acute Alcoholism					1			1	
VI.	Nervous System (Multiple Sclerosis 1 Paralysis Agitans 2)				1	1	1		3	
VIII.	Amyloidosis – Lungs					1			1	
IX.	Digestive System a. Ulcer — Gastric (Postop.)					1			1	
	Ulcer — Duodenal (Hemorrhage)				1	1			2	
	b. Cirrhosis of Liver (Alcoholism 2)		1	2	3				6	
	c. Strangulated Hernia					1			1	
X.	Genitourinary System				1				1	
XI.	Pregnancy (Hemorrhage)	1							1	
	TOTAL	1	2	2	6	6	1		18	3.5
DEATHS—All Causes		11	16	44	102	145	137	63	518	
	%	2.1	3.1	8.5	19.7	28.0	26.4	12.2		

The classification shown in Table 4 has been of great help in giving us a proper background for our selection problems in this field and the circulatory public health problem in general. The figures given for congenital and infectious heart disease are probably not comparable with those in the general population, as applicants with these conditions can seldom be accepted for insurance. The figures which record the deaths from coronary, cerebral and renal arteriosclerosis and the age groups in which they appear are representative and compel our attention.

Time will not permit us here to enter into a discussion of those conditions which some think are conducive to early arteriosclerotic changes: obesity, hypertension, and heredity. Nor can we take the time to discuss the many clinical, x-ray, electrocardiographic and other manifestations of progressive arteriosclerosis. We can refer here to only two facts which our studies and recent experiences have brought to our attention.

It seems to be quite generally accepted that early arteriosclerosis has invariably been preceded by a significant degree of hypertension. Our figures do not support this view. Although these figures may be influenced by the fact that applicants with established hypertension are not accepted for insurance, they do show that many die of advanced arteriosclerosis without evidence of previous hypertension. The sudden death of a policyholder from arteriosclerosis, who has shortly before been issued insurance with no record of hypertension, also drives this fact home to those of us who have had this experience.

The second fact which needs to be emphasized here is that arteriosclerotic changes can develop to a marked degree before they give rise to any subjective symptoms other than the usual limitations of age, or to any objective clinical, x-ray, electrocardiographic or other signs. The following cases bring this fact forcibly to our attention:

CASE 3

APPLICANT:	MALE	AGE 47
HISTORY AND EXAMINATION:	Negative	
ECG AND X-RAY:	Negative	
POLICY:	$50,000.	
ISSUED:	18 May'47	
DEATH:	20 September'47	
CAUSE OF DEATH:	CORONARY OCCLUSION	

This insured had been studied thoroughly, prior to issue of insurance, with negative results including chest x-ray and electrocardiogram. Investigations before and after death re-

vealed no limitation of business or other activities. Coronary occlusion caused sudden death four months after issue of $50,000. of insurance.

CASE 4

APPLICANT:	MALE AGE 51
HISTORY:	Negative except "Diabetes — well controlled" (Attending Physician)
EXAMINATION:	Negative
ECG AND X-RAY:	Negative
POLICY:	$100,000.
ISSUED:	7 January'48
DEATH:	20 January'48
CAUSE OF DEATH:	CORONARY OCCLUSION

The investigations of this applicant were likewise very thorough and altogether negative including chest x-ray (Figure 1) and electrocardiogram (Figure 2), except for a five-year record of diabetes well controlled under the direction of a diabetic specialist. He was issued $100,000. of insurance with the usual increase in premium covering the added risk of controlled diabetes. He fell over dead at work within two weeks after the issue of insurance.

As this case was subject to review by the local Medical Examiner, we were fortunate in securing an autopsy. The results were instructive. The pathologist reported: "the coronaries show considerable sclerosis especially the descending branch of the left where the lumen of the vessel is narrowed to pin sized caliber by atheromatous change so marked that it was difficult to cut with a knife. (Figure 3) The myocardium, however, everywhere appeared intact both grossly and microscopically."

We can only conclude from these findings that the insured died of a coronary arteriosclerosis which suddenly shut off the blood supply to the heart which itself had not yet become diseased. The fact that the heart muscle was intact probably explains why there had been no unusual symptoms or signs of

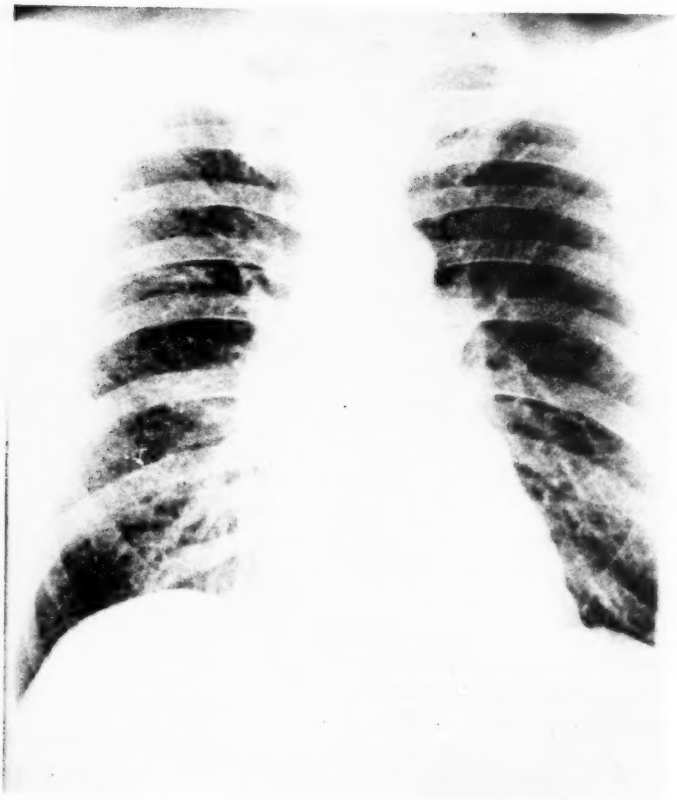

FIGURE 1

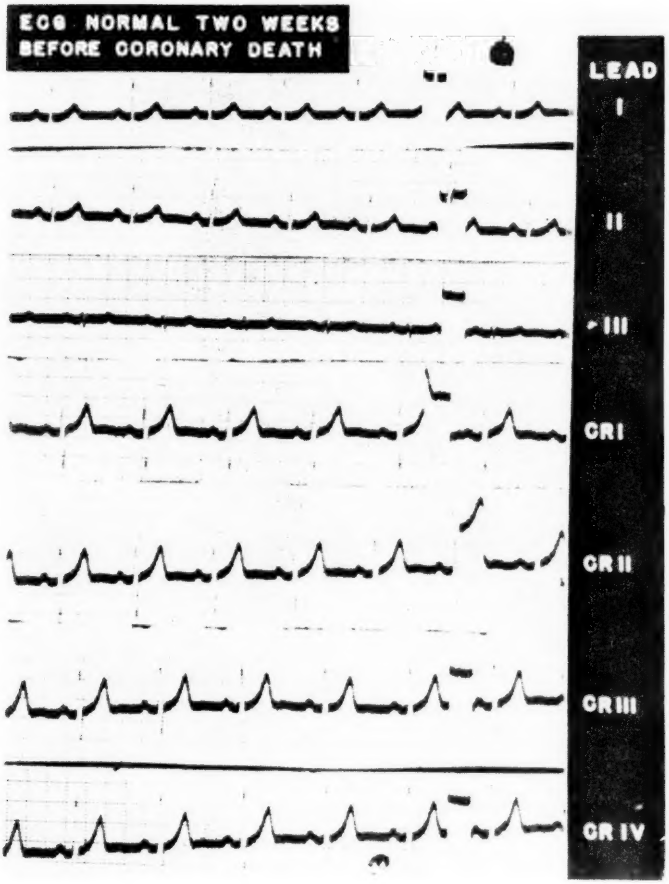
ECG NORMAL TWO WEEKS
BEFORE CORONARY DEATH
LEAD
I
II
III
CR I
CR II
CR III
CR IV

FIGURE 2

FIGURE 3

heart disease. It also explains why brilliant minds can without warning be cut down by a cerebral occlusion and why an athlete at the peak of his physical fitness can die from a coronary occlusion.

Death losses in larger or smaller amounts due to unrecognized arteriosclerotic changes will continue to occur and will undoubtedly be on the increase in the future. Our actuaries will have to tell us how many of these losses we can absorb without endangering our financial position. In the meantime, we as medical directors will be responsible to the company which we individually represent for conducting our medical investigations prior to the issue of insurance in such a manner that there can be no criticism of our acceptance of the applicant. Some may inquire why we continue with special examinations, chest x-ray and electrocardiograms, when they may show no evidence of even well advanced disease. Obviously our practice can not be determined by exceptional cases and must rather rest on the contribution to accurate diagnosis which these special studies routinely afford.

In view of the frequency of degenerative disease in this upper age group, we have recommended to our company that we raise the standard of our medical investigation of applicants over age 40, (1) by having medical examinations made whenever possible at the office of our chief examiner, (2) by calling for, whenever the amount or circumstances indicate, a chest x-ray and electrocardiogram, (3) by using these special studies instead of the second of the so called double examination which seldom yields any additional information when we use a competent examiner originally, and (4) by requiring attending physician statements almost routinely in this age group.

We hope that our Medical Directors Association will consider the problem of sufficient importance to study the situation and present recommendations which we may all consider and which will be designed to raise the standards of our medical investigation of applicants in the upper age group. It is discouraging when one company tries to carry out reasonable requirements such as those we have referred to above, only to be told by an applicant that he can secure his insurance

without complying with what most of us regard as minimal requirements.

Only a few days ago we received an application for $75,000. from a fifty-seven year old industrial executive and were told that the applicant would have to be examined in the workshop of a downtown factory. We requested that the applicant be examined at our medical office and that the examination include special studies. The applicant secured his insurance elsewhere. Few if any of us care to be faced with a large early death claim following such a makeshift medical investigation.

The problems involved with the degenerative diseases themselves are beyond the capacity of any one of us to solve. We are glad, therefore, that the companies we represent have had the foresight to establish and continue to support The Life Insurance Medical Research Fund and to place this work in the hands of the most capable medical research men in the country. We are happy too that The American Heart Association is now receiving sufficient financial support from the public to enable this organization to cooperate in this endeavor and that their work is also in the hands of leaders as eminent in this field as our speaker who is president of The American Heart Association. It has been a pleasure for us all to hear you, Dr. Harrison, and for me to join with you in this discussion of Degenerative Diseases.

PRESIDENT ROBINSON—This is a very timely and important subject. It is now open for general discussion.

DR. BERTHOLD T. D. SCHWARZ—Mr. President, I am sure that all of us here enjoyed the very informative discussions of both Dr. Harrison and Dr. Ylvisaker. I should like, however, to have Dr. Harrison clarify an apparent contradiction between two statements that he made; one, that in his practice he disregards positive findings on the electrocardiogram in favor of the evaluation of symptoms and signs. However, he apparently gives validity to the findings of the Philadelphia investigator, who made electrocardiographic studies and ten years later found that some of the findings in the electrocardiograms had prognostic value.

PRESIDENT ROBINSON—Are there any other questions?

DR. ALBERT SEATON—Mr. President, I would like to ask if in the doctor's experience he has ever been able to reduce the cholesterol content of the blood by diet.

PRESIDENT ROBINSON—Are there any other questions? Dr. Harrison covered a great amount of territory this morning. There must be some problems still unsolved in the minds of the medical directors.

DR. KARL W. ANDERSON—Mr. President, in my vicinity, we use a great deal of animal products in the form of butter and eggs and cheese; that is, in Minnesota, Wisconsin, Illinois, and Iowa. I was wondering if Dr. Harrison could tell us anything about the incidence of coronary artery disease among the farmers. It seems to me it should be higher.

DR. ARTHUR E. PARKS—Mr. President, I would like to ask Dr. Harrison if it makes any difference what time of the day a blood cholesterol is taken. In the event that it might become possible at some time to determine blood cholesterol in everyone over forty, I think that would be an important thing to know.

DR. PAUL H. LANGNER, JR.—Mr. President, I would like to ask Dr. Harrison what criteria he uses for a positive electrocardiographic test after exercise, and what per cent of positive correlation he finds between what he considers significant changes in the post-exercise electrocardiogram.

PRESIDENT ROBINSON—Are there any other questions?

DR. HARRY E. UNGERLEIDER—I would like to ask Dr. Harrison whether the electrocardiogram is taken immediately after exercise, or does he wait ten or fifteen minutes to see whether there are any findings?

DR. WILLIAM M. GENTHNER—Mr. President, this is the first talk I have heard along these lines in which the question of tobacco has not been brought up. I wonder whether Dr. Harrison deliberately avoided that or whether he thinks it is of no particular significance. We have heard so much about cigarettes in coronary sclerosis that I would like to know what he thinks about it.

DR. JOHN E. BOLAND—Mr. President, I would like to have Dr. Harrison discuss the methods of cholesterol evaluation and the approximate expense to the life insurance companies.

PRESIDENT ROBINSON—Are there any further questions?

DR. HARRISON—I cannot answer all the questions asked, but I will do the best I can with some of them. As to the apparent contradiction, what I said was that the ballistocardiographic studies of Isaac Starr in Philadelphia, a method which studies cardiac contraction and not cardiac conduction, had shown very striking correlations on a ten-year follow-up. I did not say that about the electrocardiogram. I did not mean to give the impression that one disregards the electrocardiogram. One cannot disregard anything concerning a patient. The point I wish to make is that a reliable history is worth a great deal more than all of the other information together.

I do not think electrocardiographic change should ever be disregarded, any more than fever and white blood counts, but the modern tendency is to think that an electrocardiogram tells all about a man's heart, and that modern tendency is doing a tremendous amount of harm in medicine.

Now, about the frequency of coronary disease among farmers, we have two factors to consider. I think there is some evidence to show that an outdoor life reduces the tendency to develop coronary disease. Physical activity also does this. Although not conclusive, the clinical evidence of coronary disease by occupations seems to indicate that the farmer is somewhat less apt to develop it. Necropsy reports on the incidence of coronary sclerosis, regardless of what the patient died of, conducted in New York by Dr. Levy, did not show any difference in various occupations. And yet, I think all of us have the impression that angina pectoris and myocardial infarction are more common in people in the higher income groups.

There are some figures from Denmark which would suggest that coronary disease is very common there. There are some figures with regard to the incidence in England, suggesting that it is very common there, much more common than in

Scotland. A comparison, not so much of the farmer with other groups, but of a given occupational group in different parts of the country, during periods of different economic stress, might be valuable. For instance, the farmer in certain parts of the south, who is a cotton sharecropper, eats less of milk and eggs because he cannot afford them.

We need more information about diets and their possible relation to causes of death. Certainly, it would be of very great interest to know if the farmer in the midwest, whose diet is composed of large amounts of dairy products, has more coronary sclerosis than, perhaps, the New England farmer, or the southern farmer.

I correlate the electrocardiograms taken after exercise with the degree of dyspnea as compared to a normal individual of the same age, weight, height, exercise habits, and by the presence or absence of pain or discomfort other than shortness of breath. The severity of the exercise would, of course, vary according to the age of the patient. However, I have had people show T wave changes with fright, and they can be produced with many types of drugs. To rely on the electrocardiographic criteria, the changes should be very striking. I would say that displacement of the S–T segment up or down with violent exercise, 5 millimeters or more, means coronary disease probably, provided one can exclude emotional factors, some drugs, and so on. In regard to Dr. Ungerleider's question, the duration of the change is, of course, important. If it reverts to normal quickly, it means less than if it persists.

Someone always asks about tobacco, and I never raise the question because I do not know the answer.

As to the reduction of blood cholesterol by diet, I cannot say much about that from personal experience. I see private patients only with other physicians, so I do not have a chance to follow any except those on the wards. Hence, the patients I see are not on high cholesterol diets because they cannot afford them, usually. However, judging from the studies of Tannhauser and others on people with somewhat elevated

cholesterol, one can produce reduction of slight to moderate degree in blood cholesterol in a fair percentage of patients.

I have always taken the blood cholesterol fasting. It is my impression that the cholesterol increases after a high cholesterol diet much less in proportion than the blood sugar. Figures on the fasting and non-fasting cholesterol values are available in the literature. There are several analytical methods. The ones that can be put in the hands of an ordinary technician have technical pitfalls. Some methods are highly accurate, but they require the supervision of a well trained chemist. The ordinary ones used in most hospital laboratories are not entirely accurate, although they reveal gross changes. That is one of the obstacles retarding research that would permit a decision about the effective diet for people with normal blood cholesterol. One should have the same technician, in the same laboratory, under the same conditions, performing the test to tell whether a change of 20 or 30 mg. per cent means anything.

It is my impression that with the ordinary technician these tests can be run in a commercial laboratory at a cost of five or ten dollars a test. In a hospital laboratory, doing a great many of them, the cost is somewhat reduced. I think this problem is one to which the life insurance companies could contribute information. I believe there are simple methods of preservation that would permit shipment of the blood to a central office where tests could be done by the same technician.

PRESIDENT ROBINSON—Dr. Harrison, this Association is deeply grateful to you for your contribution.

The first speaker on our symposium on atomic energy is Mr. Herbert M. Parker. After six years of research in radiologic physics at the Holt Radium Institute, Manchester, England, Mr. Parker came to the United States as a physicist to the Tumor Institute of the Swedish Hospital, Seattle, in 1938. In 1942 he became associated with the atomic energy program as a health physicist at the Metallurgical Laboratory, Chicago. He was later in charge of health physics at the Clinton Laboratories, Oak Ridge, Tennessee, prior to assuming a similar

position at the Hanford Works, Washington, which he occupies at present. Mr. Parker will discuss the "Insurability of Atomic Energy Workers". It affords me great pleasure to present to you Mr. Herbert M. Parker.

INSURABILITY OF ATOMIC ENERGY WORKERS*

H. M. PARKER

General Electric Company, Richland, Washington

Introduction

The purpose of this contribution is to describe the facts concerning life expectancy of Atomic Energy workers, in the light of the best present knowledge—to discuss the estimated validity of such knowledge—to remove hysteria on the one hand and to suggest legitimate areas of safeguards for insurance writers on the other.

The familiar mushroom cloud of the atomic bomb explosions has become a universally recognized symbol of Atomic Energy, and this recognition has to some extent been a disservice to the national interest in the field of atomic energy. The popular press has drawn so much attention to the spectacular effects of radiations, as in the wholesale destruction caused by the atomic bomb, that the people are in danger of believing that each and every laboratory experiment in the Atomic Energy Commission Laboratories carries with it somewhat the same radiation hazard. It will be shown that work in such laboratories or factories is almost certainly as safe or safer than work in other similar laboratories or factories entirely free from radiation operations. It will be shown, too, that the hazards to which Atomic Energy workers are exposed are, by and large, *not* different from those experienced by x-ray and radium workers for many years. As such workers are now generally free from extra-premium insurance, it will follow that Atomic Energy workers likewise should be good risks at regular premium for related occupations—a radiation chemical engineer at the rate for a general chemical engineer—a radiobiologist at the rate for a general biologist, and so on.

*Acknowledgement**

The author is indebted to the Atomic Energy Commission and to the General Electric Company Nucleonics Department for permission to prepare and publish data in this paper.

Consider first the adverse effects of radiation. The argument is restricted to the life insurance problem, and this is a relatively simple case. It is simple because each sample in the problem has a finite and universally recognizable endpoint —death of an individual. If one had to consider health insurance, and try to decide whether a specific malaise was or was not expedited by radiation exposure one might have legitimate areas of uncertainty. If one had to insure against a possible malformation or weakness of the second and later generations of offspring of radiation workers, one might find it impossible to write down the numerical odds. This is a problem for the successors of present day Health Physicists and Life Insurance Medical Directors. For the present problem one does not even have to decide whether a given death was due to radiation—did this man who died from leukemia have that fatal disease in any way initiated by radiation exposure? One needs only to determine what, if any, is the statistical change in life expectancy due to radiation work —what is the increment in incidence of leukemia and so on?

A. *Factors Possibly Decreasing Life Expectancy*

In the general study of radiation effects on humans or animals it is customary to divide the subject rather sharply into (1) acute exposures, usually with one or only a few large doses of radiation, (2) chronic exposures, with repeated small doses, the overall protraction of dose being largely determined by the patience and/or laboratory facilities of the observer. With death of the subject as the criterion, a different classification would be preferable. For example, in the acute exposure class, if the subject dies of a recognizable radiation injury in a finite time, the experiment is successful in contributing to an actuarial table of death-rate versus radiation dose. If, however, the dose proves to be sub-acute or leads to acute injury less severe than death we are left with a subject apparently well who may ultimately die before his normal time. This is just the subject of prime interest in the present problem, especially if he is statistically much more common than the one who has suffered a clearly labeled radiation death.

The chronic exposure class, if well protracted, will lead directly to the information sought. However, if it can be demonstrated that a feasible radiation injury is *shortening of life,* in the absence of any other demonstrable effect, or of a correlation index, no experiment is fully useful for the present purpose unless carried out over the life span of the subjects. By a correlation index one means such an index as lowering of the normal blood count by 'x' per cent to produce a shortening of life of 'y' per cent. Then, if 'y' is a single valued function of 'x', this function can be found experimentally from measured values of 'x', and the insurance problem answered.

Radiation effects will be considered under the classical headings of acute and chronic exposure, but with attempted reclassification to suit the life insurance problem.

1. *Acute Exposure of a Single Subject or Small Group*

In biological experimentation on the lethal effect of radiation on animals a mortality curve showing the per cent mortality, or alternatively per cent survival, as a function of dose is the customary primary data. The traditional mortality curve is a sigmoid curve as shown in Figure 1.

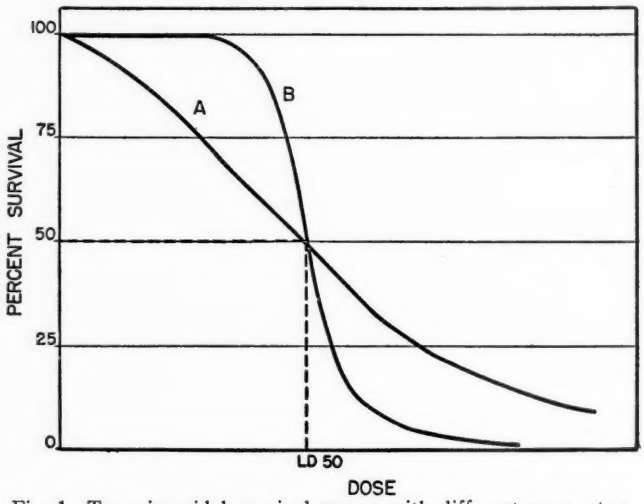

Fig. 1 Two sigmoidal survival curves with different parameters, but the same LD50

It is standard practice to use as a simplifying index that dose which kills 50 per cent of the animals. This is one kind of mean lethal dose and is written LD50. In the irradiation of a complex organism such a whole mammal one always finds a high order sigmoid curve such as Curve B. In such a case, the LD50 is a good index of lethal dose. Doses significantly less than the LD50 do not kill the animal—doses significantly above the LD50 will certainly kill the animal. Representative values of LD50 in mammals are:

Mice	— 450 — 1100 roentgens	—depends markedly on strain.
Rats	— 600 — 750 roentgens	
Guinea Pigs	— Approx. 200 roentgens	for 'x' or gamma radiation.
Rabbits	— Approx. 800 roentgens	
Dogs	— Approx. 300 roentgens	

It is believed that the LD50 in man is about 500 roentgens, and this is substantiated by some early whole body x-ray therapy treatments, and apparently by the atomic bombing experience in Japan. * To be conservative, 300 roentgens, or its equivalent in mixed radiations may be taken as the dangerous acute exposure in man. The chance of such an exposure occurring to a single factory or laboratory worker has to be determined. It is known that two such deaths occurred in the Los Alamos Laboratory during the initial pressure of work there. Beyond the fact that this proves that radiation death is possible in atomic energy manipulations, it appears proper to disregard the incidents in a statistical survey of future expectations. Typical ways in which a large exposure could occur are:

a. Direct exposure to radiation beams from nuclear reactors, cyclotrons, other particle accelerators, x-ray machines, or concentrated radioisotope sources: None of these pieces of equipment is intrinsically more dangerous than the familiar x-ray machines, many of which give dosage-rates of 300 roentgens per minute. So, in well-known fields of radiation protection, a man must not be in the

* A recent private communication indicates some British evidence that the value maybe as low as 150 r, if the dose is truly instantaneous. The variation of LD50 with time of exposure is not considered in this review. In general, the shorter the exposure time the greater the damage for a given dose. However, the order of magnitude is unchanged, and conclusions drawn here will not be affected whether one takes 150 r, 300 r, or 500 r, as the LD50 in man.

beam for a period approaching one minute. Therefore, conditions are established so that he is never in the primary beam at all. For this kind of protection it is clearly immaterial whether the new piece of equipment gives 10 or 1,000 or 1,000,000 times the dose-rate of the present x-ray machines.

b. Malfunctioning of nuclear equipment must be a theoretically existing hazard in the operation of any nuclear reactor. There is published evidence that about a dozen nuclear reactors of almost as many different types have been successfully operated. With the above-mentioned possible exception there has been no reported death in connection with these operations. It seems to be impossible to assign a numerical coefficent to this risk. There will always be some chance of malfunctioning in a newly designed apparatus of any kind. It would be reasonable to expect the risk to be steadily diminishing as more and more types are utilized.

The dangerous acute whole body dose of one hundred to several hundred roentgens is large compared with the actual acute exposures occurring in the field of atomic energy. At the Hanford Works, for example, there has to date been no single whole body exposure substantially in excess of that received in a routine radiographic examination of the chest. This amounts to about 2 roentgens, and the highest exposures have been:

1945	2.4 roentgens
1946	0.6 roentgens
1947	1.3 roentgens

There have been two cases of exposure of limited parts of the body at about 5 roentgens. Such a dose is much smaller than a typical fluoroscopic exposure of a much larger part of the body.

In practical terms, individual acute exposures are insignificant in well-organized radiation work.

2. *Major Catastrophe*

The art of nuclear reactor operation has been so shrouded in mystery as a result of necessary security that the public

is inclined to picture the nuclear reactor as a volcano that might blow its top at any time with the violence of from two to twenty thousand atomic bombs combined. In the light of present day knowledge, such an event is absolutely impossible. The nuclear reactor is operated under what has elsewhere been called the Triple Safeguard Philosophy. The unit is protected by multiple independent safety controls, and the chance of failure of all systems is perhaps about the same as that of jamming the throttle fully open, the ignition switch on, and the brakes off, in an automobile on a downgrade. If the worst did happen to a nuclear reactor, one would still not have an atomic explosion, and one might pull out of serious trouble just as the aforementioned jalopy could run out of gas and stall before it hit the wall at the bottom of the hill. However, it has been conceded in the previous section that there could be disruptive malfunctioning of a unit. Such an event could kill the operating crew of the unit, but would not be expected to kill people in nearby buildings.

3. *Chronic Exposure*

Personnel in radiation work may be exposed to repeated small doses of radiation from the following sources:

External Radiation		**Internal Emitters**
Gamma Rays	Protons	Beta Rays
X-rays	Neutrons	Alpha Rays
Beta Rays	Alpha Rays	Gamma Rays

Of these radiations, it is believed that there is sound information on the hazards arising from x-rays and gamma rays. There is a considerable body of experience in this field from industrial radiography and medical applications of these agents. In the past twenty years, the permissible exposure to such radiations has been changed only by a factor of about four. Half of this change was effected in the last few months, and was based on a growing conservatism rather than on positive findings of injury in exposed personnel. The revised permissible exposure is 300 milliroentgens per week, or 300 roentgens in a lifetime,

which represents 20 years work at this limit. In computing the exposure hazard of other radiations one resorts to a table of equivalence. One *rep* is that amount of ionizing radiation that liberates 93* ergs of energy per gram of tissue. This is approximately the release occasioned by one roentgen of x-radiation energy. One *rem* is that amount of ionizing radiation which gives the same potential biological damage as one roentgen of (hard) x-rays. Either physical measurements or calculation give reasonably accurate values of exposure in *rep*, but the biological equivalence unit, the *rem*, is uncertain to a factor of about two. Currently accepted values for chronic exposure are:

X-rays, gamma rays:	1 roentgen	=	1 rep = 1 rem, approx.
Beta Rays			1 rep = 1 rem
Protons			1 rep = 5 rem
Thermal Neutrons			1 rep = 5 rem
Fast Neutrons			1 rep = 10 rem
Alpha Particles			1 rep = 10 or 20 rem **

By this kind of system the exposure of personnel to mixed radiations can be measured separately in *rep*, and added up in terms of *rem*. The total permissible exposure is then 300 millirem per week.

For external radiation only, alpha particles are inconsequential because they do not penetrate the cornified layer of skin. Alpha particles, greatly accelerated in a cyclotron can damage the skin, and the same is true of protons. The beta rays affect only the superficial tissues. The hazard of chronic beta ray exposure is said to be the risk of cancer production in skin. Experience has shown that such cancer does not develop unless persistent deleterious changes (however slight) have been first produced by radiation. As a *lethal* agent, these beta radiations can be dismissed whenever there is reasonable inspection of the personnel.

One is left with the penetrating radiations—x-rays, gamma rays, and neutrons. It turns out that the neutron exposures are quite inconsequential in the irradiated popula-

* The *rep* has had values ranging from 83 to 93 ergs per gram. Such differences are inconsequential. None of the permissible exposure data is claimed to be accurate to within 20 per cent.

** Value still under discussion.

tion, because these particles arise only in strictly localized equipment which can be provided with permanent adequate shielding. The practical problem reduces the scope of significant chronic exposure for external radiations to x-rays and gamma rays only, and this brings one to the familiar ground of prior experience in industrial radiography and radiation therapy.

The chronic exposures to x-rays and gamma rays encountered in atomic energy installations are recorded with considerable reliability.

At the Hanford Works, the ten highest *annual* exposures have averaged:*

1946 — 1.50 roentgens
1947 — 1.75 roentgens

according to the record.

These readings were obtained by a method that does not measure less than 25 milliroentgens per week. It has been the practice at Hanford Works to charge the minimum detectable amount against the employee's record, so as to quote always the maximum exposure. Thus an employee with no true exposure would be charged with 1.25 roentgens per year. So the *real* exposures have certainly been less than the recorded amounts—almost certainly less than 1 roentgen per year.

By another more sensitive method, the average annual exposure of all employees admitted to so-called hazardous areas is reported. This has been:

1944 and 1945 — 0.9 roentgen
1946 — 0.6 roentgen
1947 — 0.4 roentgen

Here, also, there is an instrument error which is such that the readings, if wrong, are too high.

Under regimes similar to that at Hanford, the true average annual exposure of personnel will be less than 0.5 roentgen per year, and not more than 0.2 per cent of the personnel

* The 1945 record is omitted because the data were filed to permanent record without being abstracted in this form, not because they were worse than in subsequent years.

will receive annual exposures in excess of 1 roentgen. The permissible exposure limit, when these data were compiled, was about 30 roentgens per year. The revised limit is 15 roentgens per year, or about 30 times the observed exposure.

The classic effects of chronic over-exposure to penetrating radiation are:

(1) damage to skin
(2) damage to the reproductive organs
(3) damage to the blood-forming organs
(4) damage to bone

The third and fourth items are perhaps the critical ones. Here one sees the development of anemia and a fairly well substantiated increase in the incidence of leukemia. Radiation-induced bone sarcoma appears to be possible. However, in all these cases it is quite difficult to determine the exposure at which the effects begin, because of the natural incidence of the same diseases. In such a case, a statistical approach is necessary, and there is an inadequate body of cases with known exposure. For the present purposes, one can say that a good atomic energy laboratory will yield lower exposures than those experienced by the medical profession in radiation diagnosis and therapy. There is no additional risk premium for radiation hazard in these occupations.

Beyond the risk of development of a specific complaint possibly ascribable to radiation is that of a shortening of life without prior intervening symptoms of injury. Dr. R. Boche has recently reported some important data developed from a study of shortening of life in a variety of biological targets. *

It appears that the shortening of life, measured as a fraction of the life-span, is approximately the same for equal dose to all animals. If this proposition can be extrapolated to the human animal, this provides for the first time a reasonable basis for insurance assessment. It appears that

* Reported at the American Roentgen Ray Society meeting, Chicago, September, 1948.

the shortening of life is 10–4 life-spans per roentgen. Taking 65 years as the span, and 300 roentgens as the proposed permissible life dose, the shortening would be two years. This includes all causes such as anemia or leukemia, and not just shortening not ascribed to specific disease. Under the Hanford regime, the average shortening of life for 20 years of radiation work would be 3 *weeks*. There is no reason why atomic energy programs should not be continued at this standard or better.

4. *Internal Emitters*

In laboratory work, there is a finite chance that radioactive materials may enter the body, and by metabolic processes become fixed or relatively fixed in some specific body organ or organs. The only applicable body of prior information occurs in radium manipulations, such as the luminous dial industry. While much of this information is directly applicable, there is great complexity due to the wide range of metabolic behavior of the different isotopes, and to the different energies of their radiations. It is generally conceded that the most dangerous radioisotopes are the bone-seekers. Here there can be an effect on the blood-forming system or on the bone cells themselves. Fortunately, this is the case which is most analogous to the established radium poisoning data. If allowances can be made for the different patterns of distribution of deposited activity in the bone, and for the relative biological effectiveness of alpha rays, as in the radium case, and beta rays, as with radiostrontium, for example, the permissible deposition can be computed with enough accuracy. It is much more difficult to determine whether or not an individual is slowly accumulating dangerous amounts of radioisotopes. For deposition in bone, the body content can be estimated from the amount eliminated by urine or feces. This is sufficiently sensitive to insure safety. If material is deposited in the lung, one can postulate conditions under which it would be impossible to assay the deposited amount. This is a dangerous situation. It can be met by maintaining all work areas at all times at

insignificant concentrations of airborne radioactivity. Instrumentation for this purpose exists and can be operated at very high sensitivity. It has been demonstrated that deposition in the body can be held to insignificant levels by reasonable control methods. At Hanford, it seems to be assured that the internal emission of radiation is small compared with the already small external radiation. However, as this evidence is indirect, it is impossible to publish sustaining statistics comparable with the external exposure data.

5. *Unknown Quantities, If Any*

It has so far been assumed that all the feasible damaging radiations are known and can be measured. The pessimist may claim that mysterious unknown radiations emanate from the nuclear reactors. When one considers the accuracy of prediction of the physicists who developed nuclear reactors and atomic bombs, one can be reasonably assured that such is not the case. Furthermore, if these mysterious rays produced ionization in the body they would be readily detected by instruments. Without ionization, it is improbable that physiological damage could occur. A possibility is that non-ionizing radiation may produce molecular dissociation in tissue. The neutrino, known to be emitted in nuclear reactions, can presumably do this. However, if the effect were of any consequence, it would be detectable by biochemical or physical means, and this does not appear to be the case.

B. *Factors Increasing Life Expectancy*

Foremost among these factors is the extra medical care given to the great majority of radiation workers. Such care includes (1) Preselection of the personnel by initial employment examinations; (2) Blood counts and partial medical examinations at frequent intervals; (3) Comprehensive annual medical examinations. At Hanford Works, the Medical Division is finding no evidence whatever of radiation-induced change in these examinations. It is believed that the normal

incidence of ordinary diseases is being detected at an early stage. Early treatment will then certainly increase the life expectancy of the exposed population.

Vital statistics for Richland, the residence town of Hanford workers, show a ridiculously low death-rate, about one-half the national average. This is misleading for the present purposes because the population is abnormally young compared with normal communities. The population is too small to apply a satisfactory correction for skewness. At the present time, the average age is said to be 31 years, compared with the national average of 33 years. Presumably some of the death-rate diminution can be ascribed to the unusual medical care of this atypical community.

Another vital factor is the intensive Safety Program to be found in all major Atomic Energy Commission installations. The success of this can be measured by the lost time injury frequency record as follows:

Injuries per million man-hours 1945–1947

All chemical industries	9.5
Black powder	2.4
Fuses and detonators	1.9
High explosives	5.5
Small arms ammunition	2.1
Hanford Works	0.66

It will be noted that explosives manufacture, which in the popular concept is more hazardous than average chemical industry, shows a better accident rate. The Hanford Works, which again is supposed to be engaged in an extremely hazardous field, has a better accident rate than any of the quoted divisions of the chemical industry. In industry at large there is one death for every 124 lost-time injuries. Apply this rate to an idealized Hanford type plant which employs 10,000 men each beginning work at age 20 and working for 20 years. There will be two accidental deaths in this time, against 31 deaths in the average chemical plant of the same size. By the safety program alone the average life has been lengthened

in this simplified case by five weeks. This compensates for the 3 weeks shortening that has been computed at Hanford for the radiation hazard.

Throughout all the Atomic Energy Commission installations, for all production work, a similar calculation shows a lengthening of life of 4 weeks. For more hazardous research alone, the figure is 2 weeks. As all these occupations are relatively new, one can anticipate continued improvement of the safety record, in comparison with the average chemical industry. Within the accuracy of present predictions, any shortening of life by the deleterious action of radiation is fully compensated by the increased life expectancy due to extensive safety programs, and both amounts are trivial fractions of the normal life span.

Public fear of radiation also works to the advantage of protection from radiation. The laboratory or industrial worker is prone to develop a certain familiarity with and therefore contempt for ordinary tangible hazards. Experience so far indicates that he maintains a healthy respect for the intangible radiations.

C. *Comparison With Other Occupations*

It has been indicated that net life expectancy should be nominally greater in Atomic Energy Commission installations than in chemical industry at large. This may be a satisfactory bench mark for life insurance appraisals. Ultimately, the occupational hazard has to be broken down and applied to smaller groups. This can now be done by an intelligent detailed study by a special board, which should include experts in insurance problems, radiation effects, industrial safety, and laboratory safety.

Radiation therapy and diagnosis are other fields with which a comparison may be drawn. The hazards here are confined to external radiation by x-rays or radium rays only. Reported injuries in these fields continue to present an unsatisfactory picture. One of the larger clinics saw 40 injuries in diagnostic or technical work in 3 years. Four physicians are said to have died in 1946 as a result of x-ray accidents or complications.

One still finds cases of physicians who retire from radiation work as a result of prolonged changes in their blood picture. The data on apparent greater incidence of leukemia among physicians than in the general population is at least disturbing.

These data compare unfavorably with the evidence in the Atomic Energy Commission installations, and the difference is certainly not due alone to the shorter period of exposure in this new field. Rather is it due to the close detailed control that has been maintained, a type of control which it is difficult to apply to the individual therapist.

D. *Validity of Present Permissible Exposures*

It has been stated already that the permissible exposure to external radiations of the common types is fairly firmly established. Data on neutron exposures are as good as the gamma ray data to a factor of about 2 or 3. If beta ray exposures are considered equally damaging per rep as x-ray exposures, conclusions on the hazard must certainly be on the conservative side.

There are two principal areas of doubt. One is concerned with very high energy beams from new nuclear machines, such as the betatron or synchrotron. In this field, there is uncertainty in the proper method of biological dosimetry. A desirable feature of such beams is that they are produced in narrow cones so that it is relatively easy to keep personnel out of direct radiation. The radiation that is scattered out of the primary beam by and large tends to be closer to the types of radiation for which there is previous experience.

The second doubt is connected with the metabolism of radioisotopes in the body. While pilot experiments have been made with all the elements concerned, much remains to be done in analyzing the uptake, primary deposition, and retention, when the active material is supplied to the animal in various forms. Futhermore, the majority of the investigations are performed on animals, and precise quantitative translation to the human case may be questionable. No one is prepared to claim that permissible exposures are known within a factor of 10 in many cases. A customary policy in such

cases is to use a permissible deposition value 100 times lower than the best existing calculation.

E. *Legitimate Requirements for no Extra Premium Insurance*

This paper has attempted to show that work conducted with nuclear machines or radioactive materials is not necessarily dangerous to life. This does not mean that injury and premature death cannot occur in this field. The favorable results cited depend on close control of intrinsically dangerous working conditions. At Hanford, some 2½ per cent of the total manpower is needed for the execution of this control. Wherever the radiation protection force does not approximate this figure, there might be doubt as to the efficacy of the safeguards. Evidently, in a small research group it would be uneconomical to maintain a full-time radiation protection specialist, and the protection must be handled by the individual scientists, perhaps supplemented by consulting help. The recommendations of the National Committee on Radiation Protection for Safe Handling of Radioisotpes cover this point as follows:

> "In all cases, management specifically assumes the responsibility for the proper selection and maintenance of the standards necessary for safe operation. The small laboratory, handling low levels of radioactivity may modify or omit some of the following recommendations. A periodic review of such modifications by a competent radiation protection authority may be desirable. The large laboratories and industries will require more detailed control. The employment of full-time personnel qualified in radiation protection is desirable, and should be mandatory where the staff handling radioactive material exceeds 25."

Insurance organizations are entitled to satisfy themselves that standards approximating the above are established and maintained. One should look for a protection system that maintains numerical standards about ten times more rigorous than those quoted in the National Recommendations, which are to be regarded as upper permissible limits, rather than satisfactory operating levels.

The American Standards Association has successfully codified industrial radiographic installations according to the certainty with which adequate protection can be maintained. It may well be that an individual Class B installation is better operated than a Class A installation, but the probability is against this. There appears to be no reason why other laboratories or factories could not be similarly codified on the dual basis of:

1. Intrinsic nature of the radiation hazards.
2. Intensity of the protective system and safety program.

A word of caution may be in order if such surveys are instituted. A rather natural reaction of visitors to an Atomic Energy installation has been that if groups of men are required to wear personal monitoring instruments upon entry to specific areas, it is admitted that such areas are dangerous, and entrants should be subject to additional risk premium. This has the paradoxical effect of penalizing the more comprehensive protection systems. It should be obvious that a conscientious protection system should begin to apply meters to obtain factual data on exposure long before there is any finite risk of significant exposure occurring. Therefore, the survey should pay attention to the observed results from meters, and not to the geographical zones over which they are applied.

SUMMARY

The damaging effects of ionizing radiation are stated in the subdivisions of acute exposure and chronic exposure, with special reference to possible effects on the life span of the individual. Estimates of the shortening of life, based on Dr. R. Boche's theory are applied to the Hanford Works operating experience. The known operating risks for the future are indicated.

Compensating increase of life span, compared with such occupations as normal chemical manufacture or radiation therapy is expected to arise from the extensive medical and industrial safety controls applied in the field of atomic energy.

The validity of present day permissible exposure limits is discussed. Legitimate safeguards for those proposing to write insurance without extra premium are proposed.

PRESIDENT ROBINSON—Thank you very much, Mr. Parker. This paper is open for questions and discussion. Mr. Parker, if we do not have innumerable questions, I think that I might say, without being considered presumptuous by this body, that the medical directors realize where their field begins and ends. They might be a little embarrassed to enter a subject so intricate as yours is. I appreciate very much the splendid paper you have presented on this highly technical subject. It has given us vital information with respect to the occupational hazards involved.

Dr. W. Edward Chamberlain received his M. D. degree from the University of California in 1916. Following his service in World War I, he became Professor of Medicine in charge of the Department of Radiology at the Medical School of Leland Stanford University. Since 1930, he has been Professor of Radiology at the Medical School of Temple University. Dr. Chamberlain is past president of the American College of Radiology. He now holds office and membership in numerous medical and other scientific societies, including his chairmanship of the Commission on Public Health, American College of Radiology, and as consultant to the Department of Medical Research, Brookhaven Laboratory. In the field of cancer control and therapy he is special consultant to the National Cancer Institute, Bethesda, Maryland. Dr. Chamberlain's subject is "Applications of Atomic Energy to Biology and Medicine". It gives me great pleasure to introduce to you Dr. W. Edward Chamberlain.

APPLICATIONS OF ATOMIC ENERGY TO BIOLOGY AND MEDICINE

W. Edward Chamberlain, M. D.
Professor of Radiology
Medical School of Temple University, Philadelphia, Pennsylvania

I should like to begin by expressing my appreciation to Mr. Parker. It was very thrilling to me to hear his very complete, very businesslike, and very informative presentation. But he left one thing out. He did not explain to you that the marvelous record that has been produced and that he told you about, at Hanford and in other parts of the Atomic Energy Commission work, is very largely of his doing. He and a few others are responsible, but I should say that radiologists give him the most prominent place in this. It is largely his work which has resulted in these remarkable safety records.

As Mr. Parker has told you, in spite of excellent facilities, and plenty of available "know-how" for safety, there are still, today, many cases of roentgen and radium injuries. In my own practice, and within the short span of the past three years, I have seen three cases of fatal carcinoma caused by roentgen injuries to the hands of dentists who made it a practice to hold dental films in patients' mouths during x-ray exposure. These dentists were men of considerable education yet they failed to adhere to the most elementary principles of protection against this type of injury. And when they came to us for advice and treatment, after having developed unhealing ulcers on their fingers, they were apparently quite ignorant of the true significance of their lesions. Other radiologists in my community have seen yet other dentists in this predicament. And we have all seen the same frightful situation in a number of young surgeons who thought they could get away with the x-ray exposure that inevitably accompanies the setting of broken bones under fluoroscopic observation.

It is worthy of note that today these x-ray and radium injuries are practically unheard of in radiologists. This is as it should be, for the modern radiologist is a highly trained specialist and his training includes adequate attention to the technics that are necessary for safety. But in the case of physicians and surgeons who are not trained as specialists in radiology, the situation is entirely different. A little knowledge is a dangerous thing. I wish that every such physician or surgeon could hear Mr. Parker's splendid outline of the hazards of radiation and how to avoid them.

Applications of atomic energy to biology and medicine are not very new. We think of them as new because the spectacular atom bomb is so new, and the term "atomic energy" is attracting a good deal of attention these days. But ever since we began to use radium in the treatment of disease, we have been applying "atomic energy" to biology and medicine. A very large amount of energy is concentrated in every atomic nucleus. In the case of the atoms of ordinary matter, this energy is safely and permanently locked up within the unchanging, so-called "stable" nuclei. But the nuclei of certain "radioactive" atoms are "unstable". They "disintegrate" and in so doing some of their vast store of energy is released. When Becquerel, in 1898, tucked into his vest pocket some of the materials Marie and Pierre Curie were working with, he developed the now famous "Becquerel burn". This burn was caused by energy from the nuclei of disintegrating atoms of radium and the other radioactive materials that are present in the so-called uranium-radium series.

Mr. Parker is quite right in saying that all of the biologic effects of irradiation are "harmful". Fortunately we are able to use these harmful effects to advantage, by limiting the harm to those tissues or cells that we wish to harm. Quite a few of my friends are "cancer cures" through surgery; other friends of mine are "cancer cures" from irradiation. The surgeon's knife may accomplish much good, yet we must classify as "harmful" the use of a knife to cut out a part of a human being. And when irradiation produces a discernible or measurable biologic effect upon the tissues or

organs, that effect is a harmful one so far as those particular tissues or organs are concerned.

Until quite recently our sources of atomic energy were limited to the "naturally radioactive substances" like radium. But "artificial radioactivity" is becoming more and more important and we hear much these days of "radioactive isotopes". What is an isotope? What is a radioactive isotope? At the risk of inflicting boredom upon those of you who already understand these matters, I shall try to place before you an explanation of the term "isotope".

I have chosen carbon as the element which will best illustrate my points because there are five isotopes of carbon, three of them "radioactive" and the other two "stable". The "building blocks" of which atomic nuclei are made are of two kinds, "protons" and "neutrons", represented here on the blackboard as ⊕ and ○, respectively. The first thing to be said about an atom is that its chemical nature is determined by the number of *protons* in its nucleus. Protons and neutrons are practically identical in mass (weight); they differ in that protons possess unit positive charges while neutrons have no charge (are "neutral" electrically). That the chemical nature of an atom is determined by the number of protons in its nucleus is illustrated by the fact that every atom in the universe that has *six* protons in its nucleus is an atom of carbon; every atom in the universe that has *seven* protons in its nucleus is an atom of nitrogen; every atom that has *eight* protons in its nucleus is an atom of oxygen, etc.

We have said that every atom that has six protons in its nucleus is perforce an atom of carbon. Let us see how many different kinds of carbon atoms ("isotopes" of carbon) are known to exist, and how they differ from each other (see Fig. 1).

Briefly, "carbon–10", atomic weight=10 (nucleus contains six protons and four neutrons) has not been found in nature. It has been artificially produced in the cyclotron and in the "nuclear reactor" ("uranium pile"). It is extremely unstable. Its "half-life" is but 8.8 seconds. In other words, of any given

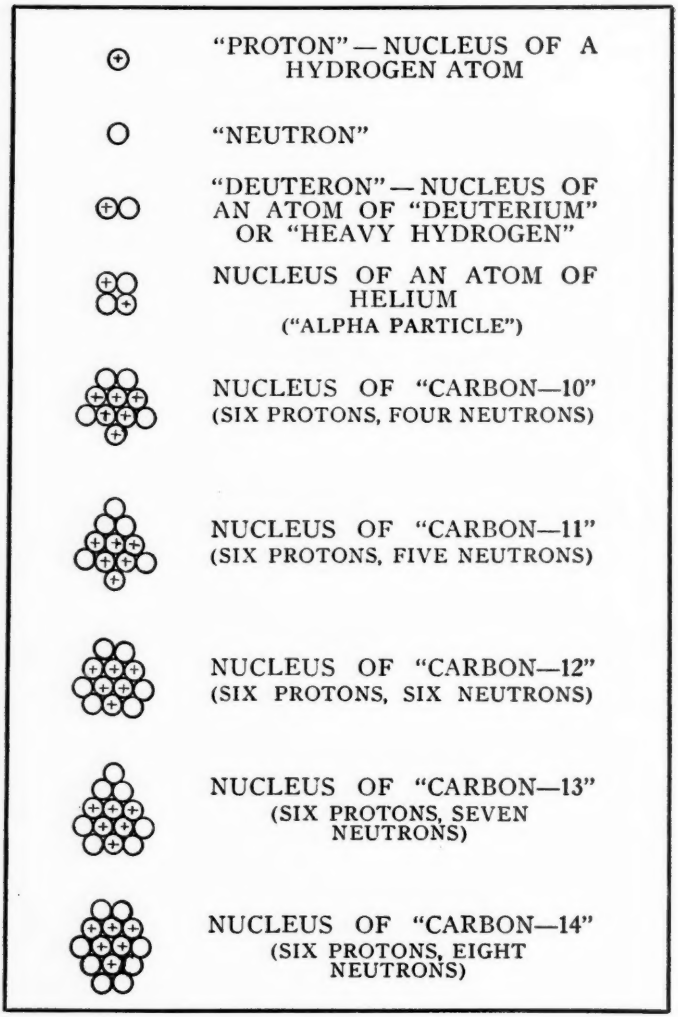

Fig. 1

The representations of atomic nuclei in this figure are but caricatures, designed to emphasize the relative numbers of protons and neutrons in the isotopes under consideration. Actually we know very little about the arrangement of the particles in the nuclei of atoms.

amount, 50 per cent has disintegrated at the end of 8.8 seconds and less than one per cent remains unchanged at the end of one minute.

Carbon–11 (six protons and five neutrons) has likewise not been found in nature but has been produced in the cyclotron. Like carbon–10 it is unstable. Its half-life is 20.5 minutes.

Carbon–12 (six protons and six neutrons) and carbon–13 (six protons and seven neutrons) are stable isotopes. As found in nature ordinary carbon is a mixture of approximately 99 per cent carbon–12 and one per cent carbon–13.

Carbon–14 (six protons and eight neutrons) is an unstable isotope which has great interest for the biologist. Its radioactivity and its convenient half-life (of the order of 1000 years or more) insure its usefulness as a tracer substance in all sorts of biochemical researches. Let us consider for a moment the possibilities that are present in such "tracer" technics.

Qualified, authorized persons may obtain carbon–14 in adequate quantities from the Atomic Energy Commission's Oak Ridge Laboratory. Organic chemists may then incorporate atoms of carbon–14 in molecules of synthetic vitamins, synthetic carbohydrates, etc. The molecules of such synthetic organic materials are then said to be "tagged" or "labeled", because they contain these little "atom bombs", the nuclei of unstable, radioactive carbon–14, and we can "trace" them as they enter into the metabolic processes that we are studying, by registering their explosive disintegrations with a Geiger counter (or other ultra-sensitive detector of radioactivity).

In any consideration of these so-called "tracer technics", one point deserves emphasis. Isotopes do not differ at all in their chemical properties. It follows from this that our tagged or labeled molecules enter into the chemical and metabolic processes that we are studying, in exactly the same way as ordinary, "normal" molecules. Because of their chemical identity with their unlabeled counterparts, Dr. Robley Evans has referred to them as "spies", successfully mingling with the general population, incapable of being identified or distinguished from ordinary individuals until they communicate,

via explosive emission of a particle or a quantum of energy and a pick-up by the Geiger counter. Let us now consider a specific instance of the use of a radioactive isotope in biologic research and medical practice.

Iodine has an important place in the physiology of a human being (or other animal). If we ingest some sodium iodide or other soluble salt of iodine, it is rapidly absorbed into the circulating blood. On each trip around in the circulation, a fraction of the blood passes through the capillaries of the thyroid gland. There the actively functioning cells of the thyroid extract a considerable part of the iodine, which thus becomes "concentrated" in the thyroid gland. It is really quite amazing how rapidly these busy thyroid cells can remove iodine atoms from the blood stream.

Now we might not be quite so sure about these facts of iodine-thyroid metabolism were it not for the ready proof we can get through the use of one of the radioactive isotopes of iodine, "iodine–131".

Iodine–131, available today in very adequate quantities from the Oak Ridge "Pile", has a half-life of 8 days, and gives out such energetic radiations when it disintegrates that accurate measurements are quite readily obtainable, even when it is diluted with some millions of parts of ordinary iodine. Suppose, for example, we give some sodium iodide, of which a very small fraction is the sodium salt of radioactive iodine–131, to a patient in whom we suspect the possibility of a hyperactive thyroid gland. Since the different kinds ("isotopes") of iodine are chemically identical, the patient's tissues and organs will treat all of the iodine atoms alike, and we can be sure that any measurement we may make of the quantity of radioactive iodine that is present, will also be a measurement of the ordinary iodine that was mixed with it at the start (since the proportions will not change).

In actual practice we save ourselves quite a bit of trouble by preparing a double dose of the labeled iodine, then dividing it into two equal halves. One-half is then kept in a bottle, as a standard, ready at any time to be compared with the patient's body by means of a Geiger counter. The radio-

activity of iodine–131 constantly decreases with the passage of time. At the end of 8 days it has decreased to one-half of its original value because one-half of all the atoms of the isotope have disintegrated. At the end of 16 days only one-quarter of the original quantity is still present. The iodine–131 in the bottle disintegrates exactly the same as that which the patient swallows. By comparing readings with the Geiger counter measuring the standard, in the bottle, with readings obtained from the patient, we can readily determine the percentage of the administered iodine that is in any particular part of the patient's body.

Now let us return to our patient, to whom the "tracer dose" of sodium iodide is to be given. At a specified hour, the patient drinks the material, dissolved in a glass of water, or in a small amount of water with a glass of water as a "chaser". Immediately after the material has been swallowed, the Geiger counter shows that all of it is in the patient's stomach, but in a remarkably short time we note evidence that the material is becoming spread out in the body, obviously through being absorbed into the blood stream. By the end of an hour we have evidence that it is quite evenly distributed throughout the body except for a very definite concentration in the thyroid gland.

At this point we begin to accumulate evidence as to the nature or disposition of the patient's thyroid gland cells. If they are normal, the observed concentration of the iodine in the thyroid will be at a very moderate rate, and when it has reached its maximum level, after about 24 hours, only 25 to 35 per cent of the total iodine administered will have become concentrated in the gland. If the activity of the patient's thyroid is *below* normal, the maximum concentration of the iodine in the gland will be abnormally low, and may be very low indeed. In extreme cases it may be as low as one or two per cent. If, on the other hand, the patient has a real "hyperthyroidism", the uptake of iodine in the gland will not only be very high, sometimes even reaching 90 or 95 per cent, but the rate at which the concentration takes place will also be enhanced. We have seen these patients concentrate as much

as 70 to 80 per cent of the iodine in their thyroid glands within the first hour after its administration.

The above-described method is without doubt the best that has yet been devised for determining whether the patient's thyroid gland is normal, hyperactive or hypoactive. But this is not all. By means of a special technic known as "radioautography", we can determine just which cells or tissues in the patient's gland are concentrating the iodine. Radioautography is particularly important when a thyroid cancer is present, for it can indicate whether or not the case is suitable for treatment with the radioactive isotope. This is how it is done.

The patient is given a "tracer dose" of the isotope, somewhat larger than the usual dose for measurement of thyroid activity. Approximately 48 hours later, the patient is operated upon and the cancerous gland either removed, as completely as possible, or a so-called "biopsy" done. (A biopsy is a removal of a small piece of the diseased gland, not for the sake of the removal, but in order that the removed piece may be studied in the laboratory.)

In the radioautograph technic, the removed tissue is prepared in the usual way, for microscopic examination, by embedding it in paraffin and cutting it into very thin "sections". When one of these thin sections has been affixed to a glass slide, it is taken into a dark room and pressed firmly against an x-ray film or an ordinary photographic film, and kept there for an appropriate length of time. (We sometimes use the Geiger counter as a sort of "exposure meter"; the greater the degree of radioactivity disclosed by the Geiger counter, the shorter the time of contact of slide and photographic film for a correct exposure.) The film is then "developed" as in the case of all ordinary photographic and x-ray "negatives", then "fixed", washed and dried. It may then be studied with the microscope and compared in detail with the tissue section from which it was exposed (see Fig. 2). The amount of blackening and its location constitute a direct record of the concentration of radioactive material in the cells or groups of cells that are easily identified on the microscopic slide.

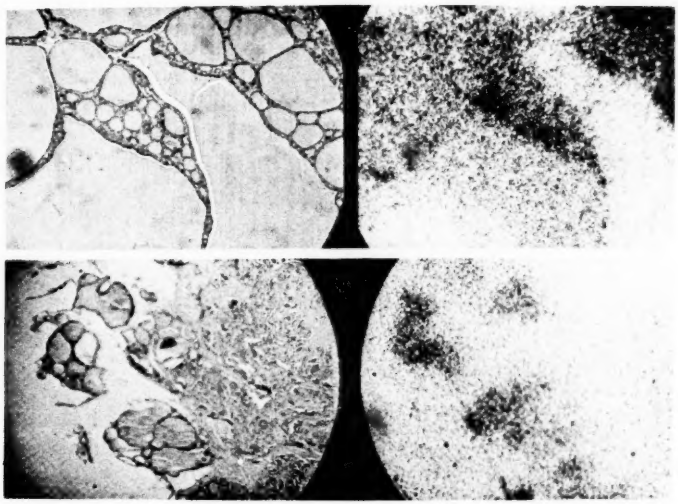

Fig. 2

Upper-Photomicrograph of a section of thyroid tissue and its radioautograph, from a patient with a non-toxic goiter. The radioautograph tells us that the labeled iodine has accumulated almost exclusively in the secretory cells and smaller alveoli, — hardly at all in the large colloid deposits (large clear spaces between the cellular elements). Lower-Photomicrograph of a section and its radioautograph, from a patient with cancer of the thyroid. The right half of the section is made up of cancer. Some islands of uninvaded and apparently normal thyroid tissue are seen on the left. The radioautograph indicates that the cancer is "non-functioning" since the uptake of iodine has been practically nil for the cancer cells.

(Taken from Hamilton, Joseph G., Radiology 39:541, 1942)

The radioautograph technic reaches its greatest importance when we use it to determine whether a particular case of cancer of the thyroid gland is a suitable one for treatment with radioactive iodine. If the cells of the cancer are "functioning" so that they exhibit an "uptake" of iodine comparable with that of the normal thyroid cells, the radioautograph will show it and we will act accordingly. By means of a mathematical formula, based in part upon a rather careful estimate of the size of the cancer, we can determine just how much iodine-131

to administer so that the cells of the cancer will receive a cancericidal (a cancer-killing) dose of irradiation.

Unfortunately such cases, favorable for treatment with iodine–131 are in the minority. The great majority of cases of thyroid cancer are like that shown in figure 2. The iodine is picked up by the normal thyroid tissues but not by the cancer. But in at least one very special type of thyroid cancer, the so-called "metastasizing struma", results have been spectacularly good. In this disease, islands of secondary cancer (we call them "metastases") develop in various parts of the body, notably in the bones. The cells of these "transplants", like those of the original tumor, are functioning. By giving the patient a tracer dose of iodine–131, and then going over his body with a Geiger counter, we can locate all of the transplants with remarkable accuracy. Then by administering a very large dose, the exact size of which will depend upon the number of metastases and their respective volumes, we may expect a most spectacular benefit to our patient.

The therapeutic value of radioactive iodine is not limited to the rare forms of cancer above referred to. It is being used increasingly as a treatment for "thyrotoxicosis" (hyperactivity of the thyroid gland; so-called "Graves' disease"). The quantity used for this condition is somewhat less than that which is necessary in treating cancer, but again the size of the dose is determined mathematically and is based upon a careful estimate of the volume of functioning tissue. (It is obvious that the dose per cell is the important consideration; the more cells there are, the more radioactive iodine must be administered.)

Another radioactive isotope with important implications for the physician and his patient is phosphorus–32. Ordinary phosphorus has an atomic weight of 31 and consists of 15 protons and 16 neutrons. In the cyclotron or in the uranium pile phosphorus nuclei containing 15 protons and 17 neutrons are readily produced, either from adding an extra neutron to a nucleus of P–31, or by a transmutation of an atom of sulphur.

Phosphorus plays an essential part in the metabolism of all living cells. It tends to become concentrated wherever

growth and new-cell formation are most active. This gives it important implications for cancer research and cancer treatment. There is reason to believe that the appetite of cells for phosphorus is in proportion to the rapidity with which they are multiplying. From this it follows that an extremely malignant tumor which is growing with great rapidity might be expected to manifest a very high uptake of phosphorus, and therefore of radioactive phosphorus (P–32). This in turn suggests the possibility of cure, for the disintegrating atoms of P–32 would, under such circumstances be located where their radioactive energy releases would do the most good. And not the least of the attractive features of such an outlook is the fact that cancers of this highest degree of malignancy are definitely incurable by other methods, such as surgery.

But there is a serious obstacle to success along the above lines. Unfortunately, for our purpose, the cells of the blood-forming tissues (bone marrow and lymph nodes) are also undergoing quite rapid multiplication and they too have a quite remarkable appetite for phosphorus. Red blood corpuscles in the average human body number approximately 30,000,000,000,000; average life of a red blood corpuscle is about 30 days; the blood-forming tissues must produce about 1,000,000,000,000 red blood corpuscles per 24-hour day. White blood cells in the average human body number approximately 30,000,000,000; average life of a white blood cell is approximately 3 days; the blood-forming tissues must produce about 10,000,000,000 white blood cells per 24-hour day. It is obvious that a human being cannot long survive a serious impairment of his blood-cell-producing function. If we give the patient enough P–32 to put an end to his tumor, we are in danger of seeing also the end of the patient. The following case history will serve to illustrate this point.

A 47-year old man developed a rapidly growing cancer which became wide-spread throughout his body even before the appearance of the first signs of disease. A piece of the tumor was removed and studied with the microscope. This revealed a cancer of a most malignant and fast-growing type.

Radioactive phosphorus was administered. To our amazement the patient began to improve almost immediately. (He had been quite ill.) By the end of the second week he was back at his place of business, insisting that he felt perfectly well. In five weeks his tumors were gone.

About two months after the administration of the isotope the tumors reappeared and grew with extreme rapidity. The patient again became very ill. Any risk seemed justified since no orthodox method of treatment offered the slightest hope. We proceeded to give him the largest amount of P–32 which we had reason to believe he could recover from. He again showed rapid improvement and soon presented a picture of well-being.

We kept the patient under close observation. In about a month he began to show a tendency to bleed, especially in the gums and under the skin, much as in the case of many of the victims of the Hiroshima and Nagasaki atom bombs. A blood count showed a very marked decrease in the numbers of "platelets" and white blood cells. We put him to bed in the hospital. A few small blood transfusions put an end to his tendency to bleed. Since the dearth of white blood cells meant a lowering of his body's defenses against infection, we gave him injections of penicillin. For more than two weeks it appeared that we might succeed in tiding him over until his bone marrow and other blood-forming tissues could again supply the missing elements. Then, suddenly, the patient died.

Autopsy showed that the cause of death was a generalized blood infection with organisms that are not sensitive to penicillin. (Streptomycin and other promising antibiotics were not available at the time.) *No tumor cells could be found anywhere in the body.*

This case illustrates very well our dilemma when we try to treat cancer with radioactive phosphorus. Even where the cancer is unusually sensitive to the radiation, a dose that is safe for the bone marrow will probably fail to cure; a dose that is large enough to get rid of the last cancer cell may cause the death of the patient.

Radioactive isotopes have important applications in the fields of physiology and pharmacology, quite aside from the diagnosis and treatment of disease. Any vitamin, food item or salt that can be synthesized, can be studied by means of these "tracer" technics, which have taught us many new facts. For example, we have recently established new concepts of iron metabolism and iron storage in the human body.

One of the most amazing of such discoveries is concerned with the metabolism of bone. The old idea, that the structure of adult bone is fairly well fixed and permanent, has had to be drastically revised. A bit of the concrete that was incorporated into the walls of the Empire State Building when that edifice was under construction, is still exactly where it was originally placed, and will stay there indefinitely, or until some fantastic accident like that airplane collision of a few years ago, displaces it. Not so with the calcium phosphate and carbonate crystals that represent the building blocks of a man's thigh bone. Tracer technics with radioactive isotopes have shown that even the most rock-like and crystalline elements, in the bones of very old people, are in a constant state of metabolic turnover. Constantly, as you and I live and eat and work, the atoms of calcium,—the molecules of the compounds of calcium that are in our bones, are being lifted out and put into solution in our body fluids and circulating blood, and chemically worked upon, and then used as the building materials for a renewal of our skeletons. Our whole concept of the metabolism of bone, of the fluidity of that metabolism, has had to be revised as a result of these studies that are made so easy for us by tracer technics with radioactive isotopes.

In the practice of medicine we sometimes have occasion to study the so-called "circulation time" of the blood, that is, the time it takes for the circulating blood to make its complete circuit, or the time it takes to traverse some specified portion of that circuit. A very good method for accomplishing this has recently been perfected. It depends upon injecting a little sodium chloride in which some of the molecules contain radioactive sodium. Strategically placed Geiger counters then tell

us just when the injected salt passes the starting point and the end point of the specified circuit. Similarly the circulation time of the sap in a tree has been measured, using labeled sodium phosphate. In one particular series of experiments, 15-foot-high specimens of the "Tree of Heaven" (the "tree that grows in Brooklyn") were studied. Geiger counters were located at the tree tops so that the arrival of the first few molecules of P–32 would be apparent. The labeled sodium phosphate, dissolved in water, was thrown upon the ground at the foot of each tree and the time of day recorded. Surprisingly enough, in two of the three trees the time was five minutes. In the third tree, which must have been sick, or at least indisposed, the time was 15 minutes. Perhaps the farmer of tomorrow will use P–32 and Geiger counters to determine which of the trees in his orchard should be culled out and replaced because they have developed some defect in their sap circulations.

Dr. Robinson, at this point I think I should offer to try to answer questions.

PRESIDENT ROBINSON—Dr. Chamberlain, it is trite to say that your lecture was marvelous. You are very kind to offer to answer questions and I am sure there will be many of them.

If you have any questions you would like Dr. Chamberlain to answer, feel free to ask them.

DR. ROBERT L. ROWLEY—Dr. Chamberlain, where does one get these extra neutrons that hitch onto these atoms?

DR. CHAMBERLAIN—A few years ago our source of neutrons for this purpose was obtained from the cyclotron in which various kinds of "atom smashing" produced a fair supply of neutrons. Today, however, we have an ever-so-much more abundant supply in the "nuclear reactor" (uranium pile). When we have a compound, or a quantity of some element which we wish to expose to an abundance of "unattached" neutrons, we have but to send it, suitably packaged, to Mr. Paul Aebersold at the Oak Ridge National Laboratory. He will arrange so that our package is placed in the "pile" where the supply of neutrons is very large. Under such circum-

stances a variety of nuclear transformations is certain to take place.

DR. ROWLEY—Well, you said that phosphorus–31 was changed to phosphorus–32. How does that come about?

DR. CHAMBERLAIN—Mr. Parker, will you please take over?

MR. PARKER—Well, the neutron glues itself on to P–31, because the neutron, being uncharged, is at liberty to wander into the structure of the nucleus without being repelled, as would be any of our charged bombarding particles that we had in the earlier classical physics. The neutron gets in around the other particles in P–31 and if the energy levels are suitable, it will stay in there and make this new "nuclide", as it is called, P–32.

What really happens is that the P–32 looks the situation over and says, "Am I better off than I was before or not?" And if it finds that really it is not any better off, then it will take steps to get rid of this imbalance in some way or other. That is why something like P–32 turns out to be a radioactive isotope. It is a less satisfactory nucleus than P–31. In 14.3 days, half of these nuclei will break up again. Now, they do not break up by just letting the same neutron escape again. That is one of the things that does not happen. It does an internal trading maneuver whereby it converts one of the particles from one form to another. If it has an extra neutron, it can convert the neutron into a proton and balance it off with a negative electron or beta particle. The beta particle is the thing that comes out of the nucleus in this case. That is how the P–32 actually breaks up. It does not go back exactly to what it was before. It trades off and makes itself into a more satisfactory nucleus.

Going to the carbons that Dr. Chamberlain mentioned, these very short-lived ones with the low total atomic number are cyclotron products. I do not remember what those particular reaction equations are, but at the other end of the scale is carbon–14, the one which, perhaps, has most hope for the future in medical applications. It can be made by allowing a neutron to be sucked up by natural carbon–13,

which occurs along with the majority member, carbon–12, in ordinary carbon.

But there is a much smarter way to do it than that; that is to take a nitrogen–14 nucleus and allow that to interact with the neutron, whereupon the things combine and a proton is eliminated. If Dr. Chamberlain would like to draw that all on the board, you will find that nitrogen–14 plus a neutron minus a proton ends up with carbon–14. There, you have made carbon–14 out of a mass of material containing nitrogen. Therefore, it is rather simple to separate the things out, and then deal with a highly concentrated form of carbon–14. That is why you end up with that instead of making it from carbon–13, because then you would have carbon–12 as 99-something per cent of the bulk of the material, and there would be most of the original carbon–13 left, plus some number of atoms of carbon–14.

This could be used as a material, but the concentrated form has considerable advantages in many of the programs that Dr. Chamberlain mentioned, on which he is actively engaged.

PRESIDENT ROBINSON—Thank you, Mr. Parker.

DR. CHAMBERLAIN—You see how we have to have the physicists at our elbows all the time, to keep us from going wrong? I am glad I let Mr.Parker make that explanation instead of trying to make it myself. Even at the beginning of the question I forgot to bring out that very important point about the neutrons being so insinuating, or insinuating themselves so easily into a nucleus because of not being charged particles.

There is a lesson in this, I think. If you are going to do anything at all along these lines, have a physicist at your elbow from early morning until late at night. That has been my formula and it has worked very well.

PRESIDENT ROBINSON—Thank you very much, Dr. Chamberlain.

Our next speaker was born in Norwalk, Connecticut. He received his B.A. degree from Fordham University and his LL.B. degree from the Law School of Yale University. For

more than fifteen years, he has occupied many important positions of public trust, including a term as Assistant Attorney General of the United States. In 1944, he was elected United States Senator from Connecticut. He is internationally known for his authorship of the McMahon Act for the Control of Atomic Energy, and now serves as a member of the Joint Congressional Committee on Atomic Energy. With such a background, he is eminently qualified to talk to us today on the subject of "The Struggle for Atomic Peace." It is a privilege and a pleasure to introduce to you the Honorable Brien McMahon, United States Senator from Connecticut.

THE STRUGGLE FOR ATOMIC PEACE

THE HONORABLE BRIEN MCMAHON
United States Senator
Connecticut

We are losing the propaganda war to Russia.

This is the most disturbing aspect of the tense world situation. Wars begin in men's minds, and only later do they merge into a war of guns, bombers, and bullets. Today the war of words—the propaganda war—is in full tilt.

Just as Hitler "softened up" his intended victims before striking, so the forces of freedom are now being weakened by an attack on their morale. As Russia wins victory after victory on the propaganda front, the danger of a shooting war looms nearer. There will be precious little hope for peace until we recapture the territory lost in men's minds.

A public opinion poll of the world would prove that the Kremlin's propagandists have half assassinated our national character. Behind the Iron Curtain, the people of Russia and Eastern Europe are being persuaded that we resemble German Nazis. In Western Europe, large percentages of the people believe that we are "war-mongers," that we are plotting an atomic attack, that we adopted the Marshall Plan for aggresive purposes, and that we itch for world empire. In Latin America, and the Orient, many millions look upon us as "imperialists."

Even in the United States itself, we have suffered propaganda defeats. Many Americans, for instance, think of our present foreign policy as a "get-tough-with-Russia" approach. This propaganda phrase implies that we have a chip on our shoulder, whereas in fact we are merely avoiding appeasement and pursuing a policy of patience and firmness.

These examples show that propaganda may often be just as successful in selling falsehood as in selling the truth. In fact, it sometimes seems that the truth is hardly more than a

drop of water in a rain barrel of propaganda. Why, then, are we losing the propaganda war? We are losing mainly for the tragic reason that Russia has converted the United Nations into a megaphone, through which she shouts charges and accusations and leads the world-wide, anti-democratic cheering section.

Our diplomats at the United Nations talk to the other diplomats—in soft, moderate tones. But Russia's Vishinsky, Molotov, Gromyko, and Malik do not talk to the other diplomats. They fire propaganda slogans over the heads of their colleagues, seeking to influence unseen mass audiences far beyond United Nations council chambers. The audacity, the brashness, and the sheer insolence of Soviet spokesmen has no precedent, not even in Hitler. They refer to their own merciless dictatorship as "democracy;" they characterize Russia, the land of slave labor and concentration camps, as the "land of freedom;" and they dare to compare America with Fascism.

These big lies reverberate daily from the United Nations forum, and thus they reach the ear of ordinary men and women throughout the earth. Mr. Paul-Henri Spaak, the distinguished Premier of Belgium, declares that the United Nations is being used as a sounding board for propaganda—instead of a meeting place for the honest settlement of differences. Winston Churchill describes the United Nations as a cockpit, in which the great powers hurl "reproaches, taunts, and recriminations" at one another.

The great injustice is that Russia exports incendiary slogans and revolutionary symbols; but she never imports opinion from the West. Russia can hit us with verbal blasts, and yet we cannot hit back—except through Voice of America radio broadcasts bootlegged behind the Iron Curtain. We are waging a war of words against an opponent who possesses a well-nigh impregnable defense. We are like a defense attorney who can only plead before part of the jury, whereas our rival can appeal to the whole jury.

Ten years ago, Mr. Vishinsky acted as prosecutor at the Moscow purge trials and few people paid any attention to

his invective because he spoke from a purely Russian platform. The practical effect of the United Nations is to give Mr. Vishinsky a world platform, from which he can pipe propaganda poison into kitchens, parlors, and living rooms in Seattle, Stockholm, and Timbuktu.

No wonder Russia is winning the propaganda war. No wonder she is delighted to attend United Nations meetings. With the United Nations as an amplifier, she can make her propaganda falsehoods echo to the Antipodes, and the Iron Curtain protects her from a Western counter-attack.

Native American communists try to use the liberties guaranteed by the United States Constitution to destroy the Constitution. In the same way, Russia uses the freedoms of the non-communist world in order to destroy those freedoms.

We all know, and have known from the beginning, that the United Nations is weak, and that it has little actual power to deal with threats against peace. But we did hope that it would appeal to world-wide public opinion and that, through world opinion, aggressors could be curbed. Our hope is turning to ashes; for United Nations debates are never reported to the more than half-billion people living behind the Iron Curtain. The United Nations is not the Town Meeting of the World—not when more than a half-billion people never find out what is said at the Town Meeting. This gross injustice, this one-way communication from the Kremlin to the rest of mankind, this lop-sided situation wherein one side can talk to the other and yet the other cannot talk back, is causing the degeneracy of the United Nations.

I know of no more tragic spectacle than that of the United Nations being used as an arena in which Soviet leaders flourish the sword of Marxian dialectics against all mankind without the slightest threat to their own powerful sway. If it cannot even serve as a World Town Meeting, if it cannot even acquaint the Russian people with both sides of the East-West debate, then it is indeed a frail instrument for peace.

Of course, the whole problem has its root in the Iron Curtain. As Mr. Hector McNeil of Great Britain has pointed

out, Russia knows to the last penny what England is spending on armaments simply by reading the English newspapers. But even the closest students of Soviet Russia do not know what she is spending on armaments. In the words of Mr. Warren Austin, American delegate to the United Nations, "Communist states have set up a closed system . . . behind which things go on in secret, things of which the rest of the world is properly suspicious. . . . So long as this situation continues, all the world will be suspicious of Soviet motives and will, of necessity, arm against unknown dangers."

As long as the Iron Curtain exists, we may as well face the fact that the threat of total war will never be lifted. The very existence of the Iron Curtain is proof that the Number One enemy of the Soviet system is TRUTH ITSELF—not the so-called imperialism of the United States, not the alleged machinations of the British Empire or of Mr. Churchill, and not the fear of a Western Bloc united for action against Russia and its satellites.

Until truth pierces the Iron Curtain, there will be no safety for Europe or for the United States or for the world. The hatred and hysteria whipped up by Soviet propaganda and Soviet deception behind the Iron Curtain may well burst forth —just as it did in the case of Germany and Japan. A people constantly misled is a dangerous people.

The average Russian is our best hope for peace. He does not want to slaughter us any more than we want to slaughter him. It is only the few ruthless inmates of the Kremlin who stand between our two peoples and who threaten "the black fury of atomic war."

Therefore, the Iron Curtain is itself an act of aggression. It should be condemned as aggression by the United Nations.

But mere condemnation will not induce the Soviet to "open the door" and let in the light of truth. Free speech, a free press, free assembly, the right to know—these are antidotes to propaganda. In a free market-place of ideas, the truth ultimately triumphs. Why should the Kremlin swallow the antidotes to propaganda, when its cynical message is flooding the earth?

Since the first explosion of the atomic bomb over Hiroshima, I have given considerable thought, especially because of my close relationship with the law to protect and develop atomic energy, to a plan through which we and the free peoples of the world might extend these freedoms behind the Iron Curtain. It is my suggestion that the United Nations should demand that the right to information, the right to unbiased news, the right to truth, be extended to all peoples. The United Nations should insist upon the right of operating its own powerful radio station in each of the Big Five nations, including Russia. It should insist also upon the right of publishing and distributing its own newspaper in each of the Big Five nations, including Russia. These activities would not be subject to Soviet interference or Soviet censorship.

Only by such concrete means can a United Nations resolution endorsing the right to know be made effective. Only by such means can the United Nations fulfill its most elementary function, that of serving as the Town Meeting of the World. The present General Assembly, now convened in Paris, has the authority to launch a truth offensive, based on United Nations-operated radio stations and newspapers. The delegates need only authorize the necessary funds and provide for a staff of expert personnel—just as the Assembly has already established a permanent headquarters staff in New York City. The Assembly can act by the vote of a two-thirds majority; it is not hampered by any veto provision.

United Nations radio stations and newspapers would publicize the viewpoint of every country. They would, for example, broadcast or print the entire speeches of the United States delegate to the Assembly, the British delegate, the French delegate, and, of course, the Soviet delegate. In this way, people everywhere—especially people behind the Iron Curtain —would hear both sides of a dispute. They would have a basis on which to make up their own minds; and the concept of a world-wide public opinion would no longer be the mockery which it is today, but a moving force which not even the most powerful dictator could ignore.

I appreciate that Mr. Molotov and Mr. Vishinsky will shake the heavens with their denunciation of this proposal for United Nations radio stations and newspapers. The Kremlin's reply will be a thunderous "no." But six months from now, when the fairness and justice of the proposal are brought home to most of the peoples of the earth, the Kremlin's "no" will sound less convincing. A year later, it will sound less convincing still. The Soviet Union will have little difficulty in thwarting the proposal so far as it applies to Russia. While there is no veto in the General Assembly, such as there is in the Security Council, the Kremlin will simply refuse to admit United Nations radio and newspaper personnel.

But the idea that the peoples of the world have a right to talk to each other, that they have a right to know, and that these rights should be enforced through the United Nations is easily understood. The proposal will appeal to the humble citizens of France and Italy and Britain, as surely as it will appeal to the sense of fairness of the American people. As soon as the United Nations expresses its will to establish radio stations and newspapers in all the Big Five countries, the Soviet word-masters will find it increasingly difficult to impress mankind with the sincerity and honesty of their motives. The democracies will immediately win a lasting victory in the propaganda war; and it may give us the opening wedge which will compel the Soviet leaders to lift the barriers against truth which separate people from people.

It would be futile to deny the practical difficulties which stand in the way of establishing a United Nations newspaper in Russia. The Soviet leaders would hardly co-operate in its distribution or permit newsboys to sell them on Moscow corners. But the principle is too vital to be abandoned. Even five copies of a United Nations newspaper passed from hand to hand through Russia would at least have an important symbolic effect.

This much we know. Until truth penetrates into the vast regions of the earth controlled by the Soviets, Russia will never agree to a reasonable solution of the problems which vex mankind. There will be no hope of reaching an agree-

ment for the effective control of atomic energy, no hope for universal disarmament, no hope for settling the problems of Germany, Korea or Austria. These will never be solved until the Russian people have the same access to the facts that our people enjoy. Only the Kremlin and its blackout on truth stand between the American people and the Russian people.

There is a precedent for this suggestion which conveys a strong hint that the Soviet leaders have a sense of guilt about the Iron Curtain. During the summer of 1946, at a meeting of the Paris Peace Conference, former Secretary of State James Byrnes directed a pointed challenge to Foreign Minister Molotov. I quote Mr. Byrnes:

> "I am entirely willing to leave the record I have read to be judged by you and by public opinion. In the United States, we have a free press. Therefore, the Soviet Representative can be sure that his charges impugning the motives of the United States have been published today in the United States press. I challenge him to secure, or permit, publication in the Soviet Union of the statements I have now made."

Even the nimble-witted Mr. Molotov was at a loss to answer these blunt and plain-spoken words of Mr. Byrnes. Within forty-eight hours, the remarks of Secretary Byrnes were quoted quite extensively in the Soviet Press, although I believe this was the first and only time in which they were.

There is a more recent indication that the Iron Curtain is far less impregnable than usually assumed. The Voice of America is being heard in Russia and there is reason to believe that it is having a salutary effect in reaching a sizable number of the Russian people. I believe the Voice of America is doing an admirable job and it may well be that Congress should expand its activities. But the peace hopes of the world are too important to rest entirely upon the slender framework of boot-leg broadcasts. Reaching the Russian people is a task primarily for the United Nations.

Under democratic procedures, government ownership of press or radio, even by the United Nations, is a poor substi-

tute for private ownership. It is our firm belief that the dissemination of news should be divorced from government supervision. But in this case there is no alternative. Russia will never print the truth in her own newspapers and she will never permit the free distribution of newspapers published in democratic countries.

Mr. Vishinsky gravely assured the world at the Paris meeting of the General Assembly that there is no such thing as the Iron Curtain. The proposal for a United Nations radio station in Russia will certainly test the validity of that statement. The world must realize the mournful fact that there will be no peace, and no genuine hope of peace, while the Iron Curtain remains. The proposal I have made rests on this simple, primary proposition—let the peoples of the world talk to the Russian people. This interchange of communication would be the greatest step for concord between nations which could be taken in our time.

A great American and a great democrat of bygone days, Thomas Jefferson, quite aptly put his finger on the dilemma which faces this modern world. Said Jefferson:

> "Were it left to me to decide whether we should have a government without newspapers, or newspapers without a government, I should not hesitate a moment to prefer the latter."

As he so often did, Jefferson spoke the language of exaggeration to impress upon his fellow countrymen a profound truth. He told us in substance that NO government will remain just and honorable unless it preserves for its people all the avenues of truth.

Here in the United States, and in other democratic countries, there is convincing evidence of the wisdom of what he said. The balancing power of truth, expressed through a free press and radio, is one of the great foundation stones of popular and decent government. Certainly, there could be no greater threat to freedom than an attempt to curb these media of expression.

The United Nations must face up to this problem. There will never be peace until positive action is taken to remove the

breeding grounds where fear and suspicion are hatched. But fear and suspicion will never vanish until all mankind is made secure in that right which underlies all other rights—THE RIGHT TO KNOW.

PRESIDENT ROBINSON—Senator, we are deeply grateful to you for this stimulating and informative address. We doubly appreciate having you with us because we recognize that our meeting comes at a time which finds your schedule crowded. At this particular time, I know that your schedule is taxed to the limit. Now, Senator McMahon has suggested that he will endeavor to answer questions, and if there are questions from the floor please do not hesitate to let us have them.

THE HONORABLE BRIEN MCMAHON—I said that particularly, Dr. Robinson, because the paper which I have read treated with the fundamental reason why we are not able to get international control of atomic energy. But, I would be most happy to answer any questions that might occur to you, either on the subject which I have discussed or on the present status of the international control plan.

PRESIDENT ROBINSON—Thank you very much, Senator.

Mr. Pearce Shepherd is not a stranger to our organization. We remember well his excellent medico-actuarial contribution upon the significance of past hypertension which he presented at the Forty-eighth Annual Meeting of our Association. He is recognized as a leader in the field of actuarial science and he has the happy faculty of making statistical information live and become understandable, even to medical directors. Mr. Shepherd will talk to us on the subject of "Pulmonary Tuberculosis Mortality—A study of Disability Claims."

PULMONARY TUBERCULOSIS MORTALITY — A STUDY OF DISABILITY CLAIMS*

PEARCE SHEPHERD
Vice President and Associate Actuary
The Prudential Insurance Company of America
Newark, New Jersey

Introduction

Interest in pulmonary tuberculosis mortality is evidenced by recent papers and discussions before your Association, the Medical Section of the American Life Convention and the American Institute of Actuaries (1).

The approach to underwriting this impairment set forth in some of these papers discarded the results of traditional impairment studies in favor of modern clinical and experimental evidence.

This paper is the result of a search for further information bearing on the problem. This is an entirely new study and we are letting the figures speak for themselves. We are pleased that the results seem to support some of our concepts of this disease. In time it may be possible to make a company or inter-company mortality study of lives insured after recovery from pulmonary tuberculosis that will reflect the recent progress in the detection and treatment of this disease; until then we must rely on statistics such as those now presented and other current information and judgment.

Description of Material

We had a group of 20,692 policyholders who had received some kind of disability benefits because of pulmonary tuber-

*Without the patient and thorough work of Mr. Preston Bassett, who directed the preparation of the statistics, this study could not have been completed. And it has been made more valuable by the helpful criticism of my medical and actuarial associates in the Prudential.

culosis. These policies had been sold by both Ordinary and Industrial agents but in every case the policy was a typical ordinary policy. In recent years new policies have provided for waiver of premium benefit only; earlier issues included a substantial number with monthly income benefits, and most of the remainder of these issues provided for the payment of the face amount of insurance over a 10 year period in addition to waiving the premiums. The policies were issued at standard rates almost without exception and did not include any lives with a previous history of pulmonary tuberculosis.

We were able to follow these lives accurately as long as their policies remained in force. We were not able to investigate many aspects that would be of interest either because the facts were not available or because they could be extracted only with great effort. Table 1 summarizes this material. Of the 20,692 lives to be observed, 12,108 had died. Some comments and explanations are appended to Table 1; other more fundamental features of the group need to be investigated before we can interpret the statistics.

TABLE 1

ORDINARY POLICYHOLDERS RECEIVING DISABILITY BENEFITS BECAUSE OF PULMONARY TUBERCULOSIS

Year in which claim became effective	Total Claims	Deaths		With-drawals	Living & Disabled 4/1/48	Living & Recovered 4/1/48
		Number	Percentage			
1919 and prior	454	386	85	68	–	–
1920 – 1929	9,053	6,989	77	1,197	122	745
1930 – 1939	7,385	3,812	52	901	388	2,284
1940 – 4/1/48	3,800	921	24	81	1,329	1,469
Total	20,692	12,108	59	2,247	1,839	4,498
1938 – 4/1/48						
Minimal	450	23	5	5	121	301
Mod. Adv.	1,642	251	15	35	574	782
Far Adv.	1,789	687	38	30	571	501
Unclassified	704	253	36	72	76	303
Sub-total	4,585	1,214	26	142	1,342	1,887

Comments and Explanations

A policyholder who had two or more policies was counted only once — that is, the experience is by lives.

Observations were measured from the date of disability, which in every case was some time after the inception of the disease; a sample showed an average of 12.8 months elapsed between the first sign of illness and the admission of the claim; for the more recent claims, however, this period was 5.7 months. In general, the date of disability was taken as the date proofs were submitted although benefits were dated back as much as a year and possibly more in some cases. In most recent years dating back did not exceed one year. Observation ceased at death or on April 1, 1948 or by withdrawal. Withdrawals occurred when the policy was surrendered or lapsed or otherwise disappeared from observation. For example, some policies provided for paying the face amount of the policy in instalments over a period of ten years and under such policies observations would cease at the end of the ten-year period as no further benefits were payable under this policy.

Complete claim files were not available for all claims beginning prior to 1938; only the claims in the latest period could, therefore, be analyzed by the extent of the disease at the beginning of claim.

We obviously do not include all the tuberculous cases in this group. We have those who were identified as having pulmonary tuberculosis, who lived long enough to qualify for benefits under their policies, and who were prevented by their illness from earning an income. We do not have those who did not live long enough to qualify for benefits or who did not let their illness prevent them from working.

In the early claims the diagnosis was sometimes based on evidence that would not be regarded as satisfactory today. It is quite likely that there were some among these early claims who never did have the disease. The mortality at long durations may be appreciably affected by this group. The evidence on later claims was more complete and usually included x-rays. A sample indicated approximately 65 per cent of these claimants had received some sanatorium treatment.

To qualify for benefits, the insured had to be totally disabled and such disability had to exist for a specified period under most policies before claim could be made. A life would not enter our study if death occurred a few months after the beginning of the disabling illness. A person who stayed disabled only a short time or who refused to stop working for more than a short time would also fall outside our study.

A sample of deaths occurring in 1947 and caused by pulmonary tuberculosis showed that only 42 per cent had been receiving disability benefits. The remaining 58 per cent was made up of 18 per cent whose illness was reported as lasting less than a year, 25 per cent whose illness was reported as lasting from one to several years, and 15 per cent where the duration was not indicated. In those cases where the illness was reported as lasting several years, it is probable that it was not continuously disabling. There is, of course, the possibility that some did not make claim because they were not aware of the benefit.

We have no information as to how many of the tuberculous persons who did not claim benefits died of other causes. There would be many with minimal disease, particularly, who would not qualify for benefits and who would die of other ailments.

Our group is nevertheless a representative group of known tuberculous lives.

The group we have studied after recovery from disability also needs some explanation. A life was considered recovered when there was medical evidence to support the company's contention that the claimant was able to work. More often recovery followed proof that the claimant was actually working and was *apparently* recovered medically. It is a well known fact that when unemployment is widespread claim rates rise and recovery becomes harder to prove. We have, however, made no attempt to measure the effect of these factors on the data under study.

The lives disappearing from observation by "withdrawal" deserve some mention. In mortality studies of insured lives it is customary to include the exposure on policies lapsing

or surrendering until that change in status takes place; the experience then reflects whatever effect such terminations have on the mortality of the persisting group and that is exactly what is desired on the assumption that such terminations will continue to take place at the same rate and for the same reasons. In the study of disability claims, the inclusion of the exposure on withdrawals is valid, but the effect on the subsequent mortality needs to be weighed in order to interpret the results properly.

Withdrawals because of maturity of an endowment, or because of the payment of the final instalment of an instalment benefit, do not indicate any selection on the part of the insured. Withdrawals by cash surrender or expiration of extended insurance indicate optimism on the part of the insured which is usually well-founded. Two-thirds of the withdrawals took place after recovery and about three-quarters of these were lapses or surrenders. There is thus reason to believe that the withdrawing group is somewhat better than those who continued to receive benefits or who continued their policies in force. In other words, if we had been able to follow the withdrawing lives until they died or until the end of observations, the death rates found would probably have been slightly lower than the death rates actually determined.

The study consists of two parts. One part concerns the mortality beginning with the admission of the claim for disability benefits and follows the life through that period of disability and through any subsequent period of recovery to death or other termination of observations. The second part of the study, more applicable to the problem of underwriting, traces observations from the termination of the first claim for disability benefits through that period of recovery until death or termination of observations in another manner.

Part 1 : *Mortality Study from Beginning of Claim*

Table 2 summarizes the experience from the date of disability. Tables 2A, 2B and 2C simply give further details in finer age and duration groups.

TABLE 2

MORTALITY AMONG ORDINARY POLICYHOLDERS RECEIVING DISABILITY BENEFITS BECAUSE OF PULMONARY TUBERCULOSIS

Period in which Claim Began and Diagnosis

Duration from Beginning of Claim in Years		Prior to 1920	1920-1929	1930-1939	1938 to 4/1/48 All (1)	Minimal	Mod. Adv.	Far Adv.
Death Rates per 1000 Lives Exposed Per Annum								
Ages 20–34	1	83	288	125	38	3*	9*	71
	2–3	188	183	85	60	14*	36	105
	4–5	185	95	45	24	6*	15	42
	6–10	96	53	28	16	3*	7*	23
	11–15	47*	23	18				
	16–20	38*	16	7*				
	21–25		17					
	26–30	18*	12*					
Ages 35–49	1	56*	334	184	90		51	127
	2–3	210	216	115	83	5*	38	137
	4–5	210	121	71	48	14*	30	76
	6–10	117	69	48	39	7*	20	44
	11–15	78*	38	34				
	16–20	103*	40	28				
	21–25	61*	32					
	26–30							
Ages 50 and over	1	125*	401	233	201		101	271
	2–3	340	319	164	136	93*	103	177
	4–5	316*	136	127	86		68	94
	6–10	37*	115	96	97	56*	92	76
	11–15	111*	75	77				
	16–20		51*	94*				
	21–25	333*	57*					
	26–30							
Actual Number of Deaths								
Ages 20–34	1	21	1476	472	87	1	8	57
	2–3	81	1185	527	223	7	54	132
	4–5	51	434	235	62	2	16	34
	6–10	37	435	301	47	1	9	20
	11–15	9	123	90				
	16–20	4	56	6				
	21–25		17					
	26–30	1	1					
Ages 35–49	1	9	932	480	140		26	83
	2–3	57	695	451	195	1	30	128
	4–5	35	254	218	72	2	16	40
	6–10	27	243	274	59	1	11	21
	11–15	9	75	82				
	16–20	6	54	10				
	21–25	2	13					
	26–30							
Ages 50 and over	1	4	292	178	117		19	72
	2–3	16	230	170	104	4	29	55
	4–5	6	52	92	38		12	15
	6–10	1	62	111	36	1	12	11
	11–15	1	20	26				
	16–20		8	3				
	21–25	1	2					
	26–30							

(1) Includes those unclassified. *Based on less than 10 deaths.

TABLE 2A

MORTALITY AMONG ORDINARY POLICYHOLDERS RECEIVING DISABILITY BENEFITS BECAUSE OF PULMONARY TUBERCULOSIS

Death Rates per 1000 Lives Exposed Per Annum

Claims Admitted, 1920–1929

Duration from Beginning of Claim in Years	Age at Disability									
	To 19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60 & up
1	391	319	278	269	318	323	385	395	427	227
2	247	242	206	198	240	267	269	315	389	412
3	233	165	145	121	146	162	209	283	286	200
4	117	102	93	118	123	133	165	77	200	
5	126	85	77	95	94	97	153	150	196	143
6	65	63	60	78	72	95	110	160	244	167
7	61	70	49	51	79	82	55	96	129	
8	44	58	62	53	70	69	50	153	111	200
9	23	33	39	44	71	44	70	49	42	500
10	12	42	35	48	41	32	96	36		
11	26	33	31	35	39	46	13	64		
12	14	12	13	18	56	15	53	68	214	
13		22	14	32	36	47	87	75	273	
14		20	18	27		34	117	56	125	
15		11	27	36	45	9	19	59		
16	33	8	26	19	36	47	61			
17	36	12	22	20	13	61	24	32	143	
18	19	18	14	9	45	80		138	200	
19		6	23	10	36	27	51	40		
20	42	17	10	12	18	50	97			
21		13	7	8	34		48	77		
22		17	18	46		59	71			
23			12	32		182		143		
24	200		18	57						
25				63	44	143				
26			50							
27										
28										

TABLE 2B

MORTALITY AMONG ORDINARY POLICYHOLDERS RECEIVING DISABILITY BENEFITS BECAUSE OF PULMONARY TUBERCULOSIS

Death Rates per 1000 Lives Exposed Per Annum

Claims Admitted, 1930-1939

Duration from Beginning of Claim in Years	Age at Disability									
	To 19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60 & up
1	77	114	127	131	155	191	226	256	233	68
2	118	112	83	108	103	145	159	170	197	148
3	67	69	65	71	81	103	125	141	182	47
4	30	45	53	53	58	82	94	134	143	220
5	32	30	44	41	52	55	117	91	97	219
6	26	27	25	35	52	77	60	131	78	130
7	35	27	30	40	36	57	58	83	75	300
8	29	28	33	31	47	35	34	85	58	
9	23	20	26	35	31	51	53	112	100	154
10	9	24	16	21	33	53	45	65	115	100
11	30	12	21	13	29	34	76	61	79	286
12	12	9	17	26	28	41	50	74	161	
13	28	21	21	32	21	40	10	50	50	
14		16	34	8	25	35		65		
15		5	13	11	32	36	87	100	100	
16		7	13		28	54	33	100		1000
17			20	12		29	67	200		
18					29					
19										
20										

TABLE 2C

MORTALITY AMONG ORDINARY POLICYHOLDERS RECEIVING DISABILITY BENEFITS BECAUSE OF PULMONARY TUBERCULOSIS

Death Rates per 1000 Lives Exposed Per Annum

Claims Admitted, 1938 to 4/1/48

Duration from Beginning of Claim in Years	Age at Disability To 19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60 & up
	All (1)									
1	25	17	32	63	67	100	116	198	219	103
2	77	56	69	76	85	96	112	134	214	200
3	71	44	49	55	50	85	80	77	127	105
4	43	26	12	34	42	52	65	145	74	125
5	26	11	21	41	27	31	85	45	43	
6	35	16	5	28	34	42	44	67	167	100
7	47	24	10	13	34	33	48	59		444
8		39	9	29	46	24	78	116	45	
9	45		14			75	40	91	83	
10		20	16	21	34	43	43	143	667	
	Minimal									
1			10							
2	77	21		14				77	222	
3		13	12	31	26				167	
4		16		19		38				
5	83						67			
6						50				
7		27						250		
8										
9										
10										
	Moderately Advanced									
1	16		9	18	39	67	50	109	98	
2		24	39	39	35	45	41	93	186	250
3	114	48	35	31	18	58	49	29	102	
4	56	11	14	22	21	37	59	130	25	
5		13	5	26	18	16	52	50	53	
6				17	34		40	34	200	500
7	59		8		15	49				
8		38	11	32				182	111	
9						56			250	
10					77		91			
	Far Advanced									
1	37	43	56	104	111	128	148	261	319	111
2	188	103	122	132	156	132	154	168	283	250
3	81	67	98	91	110	140	119	114	132	182
4	67	49	14	62	73	90	80	155	40	250
5		17	50	54	42	62	118	47	50	
6	50	10	20	58	41	55	83	111	133	
7	71	24		27	18	27	83	74		333
8		49	16		47	45	77			
9	125		24			71				
10		83		63					1000	

(1) Includes those unclassified.

The death rates, as is customary in mortality studies, are death rates from all causes; deaths from pulmonary tuberculosis, however, are obviously predominant. Where, in Table 2, durations are shown for two and five year periods, the rate indicates an average per annum rate over those durations.

Death rates per thousand per annum are shown rather than the more customary ratios of actual to expected deaths because the rates are so far above the range usually dealt with in insurance mortality studies. Furthermore, no one standard can be applied over the entire range of years covered, and a changing standard would be most confusing.

We shall point out four things shown by Table 2:

(1) Within each age group there has been a marked reduction in the mortality rate since 1920.

We know that modern methods detect evidence of the disease earlier and would expect that there would be proportionately more minimal cases in the later years. We find, however, that minimal cases constitute only 10 per cent of the disability claims approved from 1938 to 1948 (see Table 1). It would seem proper, therefore, to attribute most of the reduction in mortality to better treatment. The experience prior to 1920 is scanty and may include a larger proportion of incorrectly diagnosed cases than in the later period. The heavy first year mortality in the 1920 to 1929 period may in part be the result of liberal practices with respect to dating back benefits and allowing benefits after death had occurred.

(2) The mortality in the middle and older age groups is surprisingly heavy.

The increase in the death rate shown in Table 2 is much greater than age alone could account for. The Medical Impairment Study of 1929 (2) indicated that a personal history of pulmonary tuberculosis was of diminishing importance with increasing age and was of little importance after age 40. This was, of course, a group with a history of tuberculosis, and sometimes

a very old or doubtful history, but it has led us to regard the disease as a problem confined to young ages. It is a serious disease at all ages.

A representative group of claims at ages 50 and over was analyzed in detail. There were no cases showing tuberculosis prior to the illness for which claim was made. A few showed a probably suspicious illness a short time prior to claim. Family histories at the time of issue were negative. It may be significant, however, that one-half the group had been underweight an average of 25 pounds at the time of issue—nearly 20 years earlier. The other one-half was made up of those within 10 pounds of the average weight.

(3) The mortality decreases by duration within each age group.

The decrease is most apparent after five years. The mortality is quite high during the five year period after the beginning of the claim. Tables 2A, 2B and 2C help to show the effect of duration on mortality more clearly.

(4) The experience in the most recent period, when analyzed by degree of involvement at the time of claim, shows results in accordance with expectations, namely, that there is a lower mortality among those with minimal involvement than among those with moderately advanced disease, and a distinctly higher mortality among those classified as far advanced.

Part 2: *Mortality Among Recoveries*

The material entering into Table 3 consists of those lives that had recovered from disability sufficiently long to stop receiving benefits, i. e., they were able to resume work and were, therefore, no longer "totally disabled". They were traced in that table from the date the benefits were terminated through any subsequent period of disability until death or termination of observations. In Table 3 we are dealing with a fraction of the experience shown in Table 2.

TABLE 3

MORTALITY AMONG ORDINARY POLICYHOLDERS WHO HAD STOPPED RECEIVING DISABILITY BENEFITS BECAUSE OF RECOVERY FROM PULMONARY TUBERCULOSIS

Period in which Recovery Occurred

Duration from Recovery in Years		1920-1929	1930-1939	1938 to 4/1/48 All (1)	Minimal	Mod. Adv.	Far Adv.
Death Rates per 1000 Lives Exposed Per Annum							
Ages 20–34	1	21	4*	6*	6*	9*	
	2–3	23	11	8	4*	1*	10*
	4–5	30	16	7*			11*
	6–10	23	10	6*		4*	7*
	11–15	12	11				
	16–20	9	9*				
	21–25	4*					
	26–30						
Ages 35–49	1	16*	12	12*		10*	15*
	2–3	26	23	14		6*	20*
	4–5	31	23	13*		7*	11*
	6–10	23	21	24*		6*	13*
	11–15	24	20				
	16–20	27	14*				
	21–25	32*					
	26–30						
Ages 50 and over	1	38*	20*	24*	67*		48*
	2–3	30*	28	51	48*	39*	54*
	4–5	81*	43	14*			19*
	6–10	80	53	95*			176*
	11–15	45*	45*				
	16–20	94*					
	21–25						
	26–30						
Actual Number of Deaths							
Ages 20–34	1	15	7	6	1	4	
	2–3	30	34	13	1	1	4
	4–5	35	43	8			3
	6–10	55	57	4		1	1
	11–15	20	28				
	16–20	10	3				
	21–25	1					
	26–30						
Ages 35–49	1	6	14	9		3	3
	2–3	19	48	16		3	6
	4–5	20	44	9		2	2
	6–10	29	82	9		1	1
	11–15	21	32				
	16–20	17	3				
	21–25	4					
	26–30						
Ages 50 and over	1	2	4	4	1		3
	2–3	3	10	12	1	3	5
	4–5	7	13	2			1
	6–10	12	30	6			3
	11–15	4	8				
	16–20	5					
	21–25						
	26–30						

(1) Includes those unclassified. *Based on less than 10 deaths.

This group is much smaller than the entire group. Only 7066 lives out of our 20,692 claimants recovered from their initial period of disability to enter into this part of the study, and of that number, 975 subsequently died. (Twenty lives recovered prior to 1920 of whom 10 died; these are omitted in Table 3.) The group studied in Table 3 may be looked upon as a group of persons who might be applying for life insurance. Could they all be insured on some basis? Could they be classified into two or more classes on the basis of some characteristics with one or more of these classes insurable? Let us look at Table 3 with these questions in mind.

The death rates for those recovering in the period 1920 to 1929 are, of course, much lower than the death rates shown in Table 2 for those making claim in the same years. They are, however, five to ten or more times the death rates expected among standard lives.* There is little, if any, tendency for the rates to decrease with duration. Standard mortality does increase with duration, but the group we are studying shows a mortality substantially in excess of standard for many years after recovery.

The death rates for lives recovering in the 1930 to 1939 period are somewhat lower than those for the first period. Again, there is little, if any, improvement with duration.

The recoveries in the last period include recoveries only among those whose claims first arose in 1938 or later, whereas the two preceding groups included recoveries from any prior claims. The experience in this last group is unfortunately not large. The death rates for the last period are lower than the death rates for the preceding periods. The subdivisions of this group by the degree of involvement at the inception of the claim are small but indicate that the death rate among those with minimal or moderately advanced lesions is much better than the mortality of those with far advanced lesions

*The basic table 1920–1926 fifth policy year death rates are for comparison as follows:

Ages at issue 20–24 (attained age 27)	2.49 per 1000
Ages at issue 35–39 (attained age 42)	4.02 per 1000
Ages at issue 50–54 (attained age 57)	12.89 per 1000

even though they all progressed to the point of being "recovered". A sample of deaths occurring in a group that had been recovered from disability at least 10 years showed that 30 per cent of the deaths were attributed to tuberculosis.

Table 3 thus answers one of our questions by clearly indicating that the group of all recoveries can be divided into classes on the basis of whether the lesion was described as minimal, moderately advanced or far advanced. (The descriptions used in Table 3 applied to the lesion at the inception of the claim.)

In what other ways may the large group be divided? To assist in answering this very important question we have prepared Table 4. This may be considered a very free translation of the experience shown in Table 3 into the more familiar expression of the ratio of actual to expected deaths. Expected deaths are calculated by using the Basic Table 1925–1939 to represent standard mortality. This cannot be regarded as an entirely satisfactory table for this purpose (3) but it will do to indicate how we would expect mortality to vary by age, duration and degree of involvement.

TABLE 4

ESTIMATED RATIOS OF ACTUAL TO EXPECTED MORTALITY ON LIVES RECOVERED FROM PULMONARY TUBERCULOSIS

Years since Recovery	Minimal	Mod. Adv.	Far Adv.
	Ages 20–34		
1–2	300	500	600
3–5	200	400	500
6–10	175	300	400
11–15	150	250	350
16–20	150	200	300
	Ages 35–49		
1–2	300	400	500
3–5	200	350	450
6–10	175	250	350
11–15	150	200	300
16–20	150	150	250
	Ages 50 and over		
1–2	250	350	450
3–5	200	300	400
6–10	175	250	300
11–15	150	200	250
16–20	150	150	200

Table 4 shows these features:

1. The ratios decrease by duration.
2. They decrease slightly as we move from the youngest age group to the oldest age group.
3. The ratios for the minimal group are about one-half the ratios shown for the far advanced group. The ratios for the moderately advanced are only slightly lower than the ratios for the far advanced group.
4. There is little difference by age or degree of involvement at the longer durations.

We are going to discuss four other factors that might be incorporated in underwriting rules although our study gives very little in the way of evidence to support arguments for or against two of them.

(1) We would expect the longer the period of recovery, the better the risk would be.

We took out the experience involving those lives that had survived three years after recovery without any recurrence and compared it with the entire group of recoveries. (This was done for only the group recovering in 1930-37.) We found the death rates for the group that had been recovered for three years were 80 to 85 per cent of the death rates applicable to all recoveries.

This comparison would have been better if we had been sure that each group represented cases showing the same degree of involvement at the time of recovery. It is also possible that the groups differed by the time we got to the longer durations by having a larger proportion of incorrectly diagnosed cases in the group that recovered and stayed recovered after three years.

We investigated another aspect of this same problem by determining the rate at which recurrences of disability took place. In the first 5 years, at the younger ages, the rates were around 35 per 1000 per annum among lives recovering in the period 1920-29. The

rate was lower in 1930-39 and still lower—around 18—in the period beginning in 1938. At the older ages, recurrence rates were about 25 per 1000 per annum in all three periods. After 5 years there was a noticeable drop in the recurrence rate at all ages to around the level of 10 per 1000 per annum.

Here, too, our experience did not distinguish between groups on the basis of degree of involvement and it makes any conclusions, therefore, somewhat less reliable. If we relied on our judgment and these two tests, we would say that a waiting period of 5 years after recovery should be partially effective in getting a better than average group. At the same time we can see that if we had to make our selection on this basis alone, we could insure the group of recoveries from the beginning of recovery just about as well as we could insure them from a period three years later; we would need only slightly higher extra premiums to take this somewhat radical step.

(2) We might expect a difference in mortality between those recovering quickly as against those with a long period of disability but no greater initial involvement. An analysis of the experience by duration of disability, irrespective of other factors, showed no substantial difference. There are counteracting influences at work. Quick recoveries may mean mild illness or strong recuperative power, or, on the other hand, incomplete cure. Prolonged disability may mean more serious disease, poor healing ability, or, on the other hand, more complete and certain cure.

(3) There is strong belief that two applicants with the same other features may be distinguished on the basis of the type of lesion as shown by x-ray. That is a point on which I can throw no light whatever.

(4) Traditionally, build has been a well recognized and respected factor in underwriting personal histories of pulmonary tuberculosis almost to the exclusion of all else but duration and age. Here, again, this study

fails to add to our knowledge. We could not have gotten complete or accurate facts about build without great effort. A sample did show that disability claimants on the average had been slightly underweight at the time of issue. The departure from average was slight at ages under 40 but around 16 pounds at ages over 40.

Other Observations

One or two aspects of our investigation are not directly pertinent to either of the two principal mortality studies. They are added as a matter of interest.

For example, the groups were studied to learn something about the rate at which recovery took place. Table 5 gives this experience for two age groups. The experience for ages

TABLE 5

RECOVERIES PER 1000 PER ANNUM

Years Since Beginning of Claim	Recoveries 1920–29	Recoveries 1930–39	Claims and Recoveries 1938 to 4/1/48 All*	Minimal	Mod. Adv.	Far Adv.
Age at Disability 25–29						
1	54	76	87	171	93	33
2	95	146	200	387	212	107
3	107	183	293	507	301	224
4	99	201	292	400	315	244
5	90	188	261	273	217	206
6	91	174	228	375	177	219
7	87	192	250	333	212	235
8	92	134	218		158	227
9	111	96			77	111
10	117	113				
11–15 Avge.	86	102				
16–20 "	86					
Age at Disability 40–44						
1	39	50	83	167	86	33
2	81	93	153	322	164	80
3	102	113	195	263	240	123
4	81	101	215	176	261	150
5	64	85	175	77	216	132
6	76	83	103		103	75
7	64	64	29		42	30
8	63	71	113	167	59	167
9	52	58	138	200		250
10	54	43				
11–15 Avge.	23	58				
16–20 "	19					

* Including unclassified.

25 to 29 shows that recovery rates were higher in the thirties than they were in the twenties, and still higher in the period beginning in 1938. In this latter period the variation by degree of involvement is in accordance with expectations. It is interesting to note that even the far advanced group in this period showed better recovery rates than the entire group of claimants in either the 1920 or 1930 periods.

At ages 40 to 44 the same characteristics are shown by periods as for the younger ages. The recovery rates in this age group are uniformly somewhat lower than the corresponding rates at the younger ages. This may reflect a difference in the disease as it applies to the older ages, but it undoubtedly also reflects the economic fact that it is difficult to effect recovery from disability at the older ages after benefits have been paid for a few years.

As a matter of interest, Table 6 has been prepared to show what might be expected to be the status of 1000 cases of a certain class after 5 years, using death and recovery rates based on the experience from 1938 to 1948.

TABLE 6

STATUS OF LIVES 5 YEARS AFTER BEGINNING OF CLAIM

(Based on rates of death and recovery experienced on claims from 1938 to 4/1/48)

1000 Lives in each Age Group and in each Class by Degree of Disease

Status	All (1)	Degree of Disease Minimal	Mod. Adv.	Far Adv.
Ages 20–34				
Dead	193	49	112	314
Recovered	570	858	655	397
Still Disabled	237	93	233	289
Ages 35–49				
Dead	307	41	180	436
Recovered	438	718	561	297
Still Disabled	255	241	259	267
Ages 50 and over				
Dead	497	214	369	584
Recovered	179	473	190	133
Still Disabled	324	313	441	283

(1) Includes those unclassified.

Conclusions

The study of mortality among disability claimants throws some light on the progress made in recent years in detecting and treating this disease. It clearly indicates that pulmonary tuberculosis is a disease accompanied by a heavy mortality at any age.

Questions that arise in underwriting applicants who have had pulmonary tuberculosis cannot be positively answered by the statistics. We can, however, deduce some highly probable facts, such as (1) our underwriting of applicants at the middle and older ages has probably been more liberal than is justified, (2) the earlier the disease is discovered, the lower the mortality, (3) the longer the period of recovery, the better the mortality, and (4) some extra mortality, however, must be anticipated in practically every case even after several years without a recurrence.

REFERENCES

1. Kirkland, H. B.: Personal History and Contact in Pulmonary Tuberculosis, Medical Section, American Life Convention, Chicago, 1946, p. 20.
   Open Forum on Problems of Medical Selection, Transac. Assoc. of Life Ins. Med. Dir. of Amer., Vol. XXX, New York, Recording & Statistical Corp., 1946, p. 280.
   Fellows, Haynes Harold: Relationship of X-ray and Tuberculosis in Underwriting, and discussion, *ibid.*, Vol. XXXI, 1947, p. 95.
   Lyle, A. M.: The Selection of Tuberculosis, The Record, American Institute of Actuaries, Vol. XXXIII, No. 68, Chicago, 1944, p. 319.
   Shepherd, Pearce: Ratings for Tuberculosis, *ibid.*, p. 390.
2. Medical Impairment Study, New York, 1929, p. 104.
3. Shepherd, Pearce: Analysis of Blood Pressure Mortality, Medical Section, American Life Convention, Chicago, 1941, p. 66.
   (The comments with reference to the Basic Table 1925–37 apply equally well to the Basic Table 1925–39.)

PRESIDENT ROBINSON—The discussion of this paper will be opened by Mr. Edward A. Lew, Assistant Actuary of the Metropolitan Life Insurance Company.

MR. EDWARD A. LEW—Mr. Shepherd has performed a great service in presenting this paper. The new information it gives us about recent mortality of insured lives who recovered from pulmonary tuberculosis is particularly timely.

I believe Mr. Shepherd's paper is notable for three distinct contributions:

First, it provides us with new information about recent death and recovery rates among persons disabled by pulmonary tuberculosis. This information sheds further light on the prognosis in different stages of the disease.

Secondly, and perhaps most important, it presents data as to the recent mortality of Ordinary policyholders who recovered from pulmonary tuberculosis, and puts forward conclusions regarding the mortality which may be anticipated among such persons. These data and conclusions bear directly on the ratings for personal history of pulmonary tuberculosis.

Finally, it introduces a new method for investigating mortality of insured lives with histories of serious disabilities. This method has general application and relies on the ability of insurance companies to trace the standing of policies on which disability claims have been terminated by recovery.

Before discussing these three topics, it might be well to emphasize that the problems presented by persons suffering from tuberculosis and by those who have recovered from this disease still involve large numbers of individuals, despite the fact that the tuberculosis death rate of white persons in the general population has decreased by more than 70 per cent since 1920 (from about 100 per 100,000 in 1920 to less than 30 per 100,000 in 1946). The number of disabling cases of tuberculosis in the United States is currently estimated at about 300,000. Some 120,000 new cases of tuberculosis per year have been reported to the Public Health Service in recent years, while hospital admissions for tuberculosis totaled nearly 100,000 in 1947. Moreover, there were nearly 51,000 deaths from tuberculosis during 1946, and of these almost 47,000 were classified as due to pulmonary tuberculosis.

Mortality of Insured Lives Disabled by Tuberculosis

The first part of Mr. Shepherd's paper deals with the mortality of the Prudential's Ordinary policyholders after they were allowed waiver of premium, instalment or income dis-

ability benefits on account of pulmonary tuberculosis. This experience covered not only the period during which disability benefits were paid but also, in cases where recovery ensued, the time subsequent to termination of disability benefits as long as the life insurance policy remained in force. For comparison with this experience I had available a less extensive study of the mortality among the Metropolitan's Ordinary policyholders in receipt of disability income benefits on account of pulmonary tuberculosis during the period from 1918 to 1939 anniversaries. Only the experience during disability was covered by this study. A comparison of the Metropolitan's experience with the Prudential's corresponding experience, during disability only, brought out that the death rates of Ordinary policyholders disabled by pulmonary tuberculosis were much the same in both companies after the first year of disability; in the first year of disability the Metropolitan's death rates were considerably lower, probably reflecting the effect of the different provisions of the Metropolitan's total and permanent disability income clauses (such as longer waiting period under some clauses). A comparison of the recovery rates in the two experiences indicated that they were also in line. This agreement between the two experiences adds, of course, to their credibility.

In his discussion of the mortality among the Prudential's policyholders disabled by pulmonary tuberculosis, Mr. Shepherd first calls attention to the marked reduction in the death rates since 1920. He attributes most of this decrease in mortality to better treatment and notes that only about 10 per cent of all tuberculosis disability claims were diagnosed as minimal at the time they were admitted. In the Metropolitan's experience just referred to, practically all of the cases allowed disability income benefits were either moderately or far advanced. The very small proportion of minimal cases in both experiences is not surprising in view of the fact that the criterion for allowing disability benefits was inability to work. There is some question, however, whether better treatment of moderately and far advanced cases of tuberculosis could account for all of the improvement in mortality found

in the Prudential's experience. The Metropolitan's experience suggests that earlier detection of the disease indicated by increasing reliance on x-ray diagnosis was an important factor in this respect.

There is considerable evidence that most of the reduction in tuberculosis mortality in the general population since 1920 has been due much more to a decreasing number of new cases of tuberculosis than to a markedly lower case mortality. Specifically, the number of new cases of tuberculosis reported has declined from about 187 per 100,000 population in 1920 to 84 per 100,000 population in 1946. Thus, a 55 per cent decrease in the number of new cases per 100,000 population has been paralleled by a reduction in tuberculosis mortality in the general population of about 60 per cent (for white and colored persons combined). From these figures it would appear that the substantial decline in the number of new cases has been a more important factor in lowering tuberculosis mortality in the general population than better treatment. On the other hand, the fact that a large proportion of tuberculosis patients leave sanatoria or hospitals against medical advice makes it impossible for population mortality statistics to reflect fully the effectiveness of new methods of treatment.

Mr. Shepherd next mentions the relatively heavy mortality from tuberculosis in the middle and older age groups. The higher mortality found at these ages in the Prudential's experience for the last ten years is in line with the progression of tuberculosis death rates by age among white males in the general population. In 1946 the tuberculosis death rate of white males in the general population was 24 per 100,000 at ages 25–29, increasing to 46 per 100,000 at ages 35–44, and to 96 per 100,000 at ages 55–64. One of the reasons for the high mortality from the disease at the middle and older ages may lie in the tendency of males at these ages to continue working as long as possible. A number of sanatoria have reported that older persons are generally admitted in more advanced stages of the disease and that older men leave more frequently against medical advice.

The death rates recently experienced among veterans drawing disability pensions on account of tuberculosis highlight the point made by Mr. Shepherd that the mortality of persons suffering from it decreases with duration from date of admission. Such veterans of service in World War II are relatively young men in their late twenties and early thirties who must have contracted the disease from three to five years ago. Those drawing service-connected pension awards for tuberculosis include a substantial proportion of minimal cases and they recently experienced a death rate of 36 per 1,000; those drawing non-service-connected pension awards for tuberculosis are all totally disabled and they recently experienced a death rate of 190 per 1,000. By contrast the veterans of World War I, who are largely in their fifties and who on the average acquired tuberculosis many years ago, experienced during the same period a death rate of 15 per 1,000 in the case of those with service-connected pension awards and a death rate of about 100 per 1,000 in the case of those with non-service-connected pension awards. These figures certainly support Mr. Shepherd's conclusion "that pulmonary tuberculosis is a disease accompanied by a heavy mortality at any age."

Mortality of Insured Lives after Recovery from Tuberculosis

The remarkable decline in the mortality from tuberculosis in the general population as well as advances in methods of detection and treatment of this disease has naturally raised questions as to the propriety of present ratings for personal histories of tuberculosis. The new information adduced by Mr. Shepherd from the Prudential's experience among Ordinary policyholders who recovered from pulmonary tuberculosis should help us to answer these questions. Mr. Shepherd's interpretation of the Prudential's experience in terms of ratings is, of course, of special value to all concerned with the underwriting of life insurance risks presenting a personal history of tuberculosis.

In translating the Prudential's experience into ratings, it is necessary to take account of certain peculiarities of the data that arise from the method used in making the investi-

gation. One of these is the fact, pointed to by Mr. Shepherd, that withdrawals from the experience after recovery have probably drawn off the lives among whom the lowest mortality might have been anticipated, so that the Prudential's experience may to a degree overstate the mortality of insured lives who recovered from pulmonary tuberculosis. Another is the difference between the date of recovery assumed in the Prudential's experience and the recovery date that would probably be reported on an application for life insurance. In the Prudential's experience the date of recovery was taken as the date as of which disability benefits were terminated, whereas persons applying for life insurance would probably tend to report an earlier date as the date of recovery. Still another point to bear in mind is that the Prudential's experience of necessity distinguished between the different stages of the disease at the time the policyholder was admitted to disability benefits, whereas applicants for life insurance would probably report the degree of involvement diagnosed at the time of their recovery. In my judgment, reasonable allowance for these differences would not in any way affect the validity of Mr. Shepherd's conclusions regarding the significance of the Prudential's experience.

Based on other considerations and guided by a small experience on a group of our own employees, we in the Metropolitan reached conclusions as to the proper ratings for personal history of tuberculosis that are remarkably close to Mr. Shepherd's interpretation of the Prudential's experience. We agree with Mr. Shepherd on the emphasis he places on the degree of involvement and on the duration from recovery. We further agree that age is not a very important factor in the rating schedule.

Method Used to Investigate Mortality after Recovery from Disability

I would commend to your attention and to the attention of your actuaries the method which Mr. Shepherd used in developing the recent mortality experience among the Prudential's Ordinary policyholders after recovery from pulmonary

tuberculosis. This method can be applied equally well to investigate the mortality of Ordinary policyholders after recovery from other serious disabilities. It is likely to be particularly fruitful in companies which have written a large proportion of their Ordinary business with disability benefits. One of its major advantages lies in the opportunity it affords to review the type of case included in the study in the light of the detailed information available on disability claim papers. The findings for many histories of disability obtained in this manner could probably be interpreted with a greater degree of understanding than in the case of histories based on applicants' own statements in life insurance applications. It is, however, necessary to keep in mind that any experience based on a follow-up of disability claims would not yield results that are in all respects as pertinent to life insurance underwriting as those based on standard mortality investigations of insured lives who reported histories of disabilities on their applications.

The method used by Mr. Shepherd resembles one of the projects of the Committee on Veterans Medical Problems of the National Research Council. This Committee has been making plans for follow-up studies of veterans treated for various conditions, including many serious disabilities. In the case of veterans drawing disability pensions, the follow-up is automatic so long as the veteran remains disabled and cashes his pension checks. However, the Committee on Veterans Medical Problems has also been giving consideration to possible methods of following up veterans after recovery. If some of these methods prove successful, the Committee's findings with respect to the mortality of veterans suffering from various disabilities or impairments will undoubtedly be of value in life insurance underwriting.

PRESIDENT ROBINSON—Thank you, Mr. Lew. Discussion of this paper will be continued by your very good friend and mine, Dr. Harry W. Dingman, Vice-President and Medical Director of the Continental Assurance Company.

DR. HARRY W. DINGMAN—Mr. President, Members and Friends: The Prudential has made a series of studies on tu-

berculosis mortality. The lead-off study was made by Miss Lyle in 1945 with the completeness and clearness that characterize everything she does. Further studies were contributed in 1946 by Dr. Kirkland; in 1947 by Dr. Kessler; and now the fourth, a paper by Mr. Pearce Shepherd, is prepared with the carefulness and thoroughness that characterize everything he does.

Conclusions from these four studies are largely these: Contact is far and away the most important factor in the spreading of tuberculosis. Weight and family history are secondary factors, possibly minor factors. Age is a factor in which we have to reverse our conceptions and put the stress at the older ages rather than the younger. Mortality is in direct ratio to severity of the disease. Recurrence must be expected within five years in 15 per cent of those who appear to have made clinical recovery, and in 20 per cent in a ten-year period.

Estimate is—and here I differ with Mr. Lew—that there are one million persons with active tuberculosis in the United States, and they know it. Estimate is that there are one million persons with latent tuberculosis in the United States, and they do not know it. Estimate is, further, that there are three or four million persons, more rather than less, who are going to get tuberculosis. This very day, the 29th of October, 150 persons are dying of tuberculosis. During the time Mr. Shepherd was talking, five people died of this disease.

Economic loss is staggering—two million dollars a day, three-quarters of a billion dollars a year. This includes medical care, hospital care, nursing care, and loss of wages. Yet, it is not impossible to prevent this disease. Back in 1906 Calmette and Guerin started studying some "bugs" in the Pasteur Institute, Paris. They got very friendly with the tubercle bacilli and started feeding them potatoes and bile. The organisms were quite amenable to this treatment and were relieved of a lot of malignant propensities. These "bugs" can now be introduced into humans like you and me and produce immunity. They give immunity to those who are tuberculin-negative, a hundred million such people in the United

States. It is practical. Some ten million persons have already been vaccinated, and the evidence is that they acquire immunity for a five-year period.

So much for the general population. What about the insured population? That is where Mr. Shepherd has given us such important information to date on over twenty thousand subjects.

The insured population is in the upper economic group. They get better care and earlier diagnosis. Therefore, they should have shorter periods of disability. But this is not so. They demand higher standards of health and take longer terms of disability. They can afford this and they can let their insurance companies bear the cost.

More than any of us, Mr. Shepherd recognizes the heterogeneity of his material. Some diagnoses were made by good doctors, some by doctors not so good, some by doctors almost good. Some were made without x-ray examinations. Some were made without sputum examinations. Some were erroneous diagnoses because of pleurisy, histoplasmosis and cancer. Some were fraudulent diagnoses so that the individuals could collect pensions and retirements.

Mr. Shepherd recognizes more than most of us that weight can be an index of nutrition, and that family history can be an index of body sturdiness. But he also recognizes that the skinny fellow may be very healthy and the blemish in family history may be accidental. Therefore, he has confined his analysis very largely to age and severity.

It would be interesting to know what the average period of disability was for the persons who fell under his observations. Recently, I read in a "Public Health Report" of half a million disabled persons collecting from benefit societies. Of these, 270 or 280 persons with tuberculosis collected on the average for a period of twenty-eight months. But the maximum collection period allowed by these benefit societies was fifty-two weeks, and with some of them only twenty-six weeks. I remember, eight or ten years ago, another "Public Health Report" in which some government employees collected forty

months on an average. I would like to know how long, if you happen to have that information, Mr. Shepherd, how long these insured persons were disabled.

The practical point of the paper, as I see it, is whether or not the experience of the Prudential will now replace our older studies, including the Medico-Actuarial Mortality Investigation of 1913. This was a study of persons who lived fifty or more years ago. On basis of present investigation, we may ignore, it seems to me, the weight factor and family history. Do not forget age, but do not over-stress it; and any stress given should apply to the older ages rather than the younger. Do not forget recency of attack, but do not over-emphasize that. And do not forget degree of severity. There is a point that we overlooked in our older studies and now we know it is important.

With those factors in mind, the following conclusions would seem logical to me. Within two years of clinical recovery, appraise Table H. That is 300 per cent mortality; years 3–4–5, Table D, which is 200 per cent mortality; years 6 to 10, Table C, that is 175 per cent mortality; and after 10 years, all the way to 20 years, Table B, which is 150 per cent mortality. Double these ratings if the person had a severe case of tuberculosis.

Mr. Chairman, I feel indebted to you and to the group I am addressing for having the opportunity to discuss so splendid a paper.

PRESIDENT ROBINSON—This paper is now open for general discussion and questions.

DR. SCHWARZ—Mr. Chairman, we are indebted to our friends, the actuaries, for their very fine studies. They must, because of their vocation and profession, essentially look backward, and it is good to look backward because the data that we obtain are valid for the past. But in the medical profession, we feel that things are not static but are advancing, and I know that our actuarial friends recognize that too.

I was very glad to see Mr. Shepherd point out in his charts that the far advanced cases of tuberculosis that have recovered

showed a very striking percentage increase in number. That means something in forecasting for the future, as intimated by him, successful methods of new therapy. Taking into consideration the years over which that study has been made, we know that chest surgery has advanced considerably and that we may allocate to that branch of medicine the very notable triumphs in the reduction of mortality in the far advanced groups.

We know that in the last two years we have acquired a wonderful antibiotic drug, streptomycin. We know that it is of very great value, certainly in all cases of tuberculosis that require surgery. But we still have to determine what beneficent prognostic results will accrue from the use of streptomycin in the very early progressive tuberculosis which Hinshaw, of the Mayo Clinic, believes responds favorably. I believe, therefore, that we can take these studies made by Mr. Shepherd and forecast for the future a greater hope for a better life expectancy for tuberculous individuals.

PRESIDENT ROBINSON—Thank you, Dr. Schwarz. Are there any questions which anybody would like to ask Mr. Shepherd?

DR. HAROLD D. DELAMERE—Mr. President, I would like to ask Mr. Shepherd what, in his opinion, would be the effect on that table of mortality suggested there by a current physical examination. Would it be possible to rule out a good deal of that extra mortality at the moment?

PRESIDENT ROBINSON—Are there further questions?

MR. SHEPHERD—The question was, how much importance would a current physical examination have in taking this whole group of recovered lives and picking out the best ones. I think it would be quite a bit of help to you, although I have no way of knowing. We did not have any information along that line because we could not get it.

But I think we all realize the moderately advanced group, while they are labeled "recovered", certainly could be sorted out into classes considered better than average, worse than average, and average. One would naturally start working with the group considered better than average.

Now, what the variation is I do not know. It may be possible to select some of them that would give a standard mortality; it may be possible to select some that would not even do that. I think you would undoubtedly find that some of them would give a very heavy substandard mortality. I could not answer that in detail.

While I am up here, I would like to say that I appreciate very much the discussion of this paper by Mr. Lew and Dr. Dingman. They have both added a great deal to it. Dr. Dingman in particular has been very kind.

I would like to say this about Dr. Schwarz's comments. It is true that actuaries do look backward, but I do think they look ahead also, because we are predicting the premium rates being charged for policies that are going to be in force for the next fifty or sixty years. If he has the impression that we have had our heads turned backward for a long time, I think we could change that picture, and picture ourselves as Janus, looking both ways at once.

PRESIDENT ROBINSON—The Moderator of our Open Forum does not require an introduction to this audience. We all know him as a fine gentlemen and an able medical director. It is a pleasure to present to you Dr. Ralph R. Simmons, Medical Director of the Equitable Life Insurance Company of Iowa, who will act as Moderator.

OPEN FORUM

on

PROBLEMS OF MEDICAL SELECTION

RALPH R. SIMMONS, M. D., *Moderator*

Equitable Life Insurance Company of Iowa
Des Moines, Iowa

To introduce and preface this portion of the program, it would seem in order to state the objectives as well as the procedures concerned in this open forum. Due to interruptions of office routine by the late war, as well as by a number of unforeseeable events, up-to-date mortality studies so essential to satisfactory underwriting are not now available.

Recent advances in medical science impress each of us with the need for change in the rating of many impairments and emphasize the fact that these changes cannot be predicated satisfactorily on the Medical Impairment Study of 1929 or even the supplementary studies of 1935 or 1939. To fill the gap between these older statistical studies and those newer studies now being planned but as yet unavailable, we need a studious and frank exchange of ideas on frequently encountered medical impairments, the mortality complexion of which has been so signally changed by the newer diagnostic procedures and modern therapy.

To fill this need and to bring to your attention some well-considered suggestions on medical selection in these changing fields, your Program Committee again this year included an open forum section in the proceedings of this meeting. Your Moderator has asked each speaker to epitomize his presentation in order to provide time for questions and some discussion from the floor. You are invited to review the complete and unabridged texts of these papers in the forthcoming published Proceedings of this meeting.

In order to conclude this portion of the program in the scheduled time and yet give voice to all who wish to participate in the discussion, your Moderator requests that all questions or comments from the floor be stated as concisely as possible, to reflect adequately your specific opinion or to reflect the current practice of your company. Terseness in discussion will permit more of you to participate in this valuable phase of the program and allow time for the essayist to answer your questions.

It is now my pleasure to introduce Dr. Peter Denker as the first speaker. Among his many accomplishments is that of Associate Visiting Neuropsychiatrist, Bellevue Hospital, New York City. Dr. Denker will discuss "Some Recent Advances in Knowledge of the Epilepsies."

DR. PETER G. DENKER—The past decade has been productive of great advances in our knowledge of the epilepsies. By refinement in the sensitivity of the string galvonometer, similar to the mechanism of the electrocardiograph, the action potentials of the various brain areas can be easily recorded on the electroencephalogram, or "brain wave" test. This simple procedure which requires merely the pasting on of electrodes to the various scalp areas has been of great help in diagnosis, especially in the borderline cases of epilepsy. The normal alpha rhythm, or 10 per second waves, is considerably altered in amplitude and frequency in grand mal epilepsy, whereas in the petit mal type, the more common "spike and wave" formations are found. In this manner, many dubious "fainting" or "dizzy" spells are correctly diagnosed. By and large, positive electroencephalograms are found in 75 to 80 per cent of epileptics. It must be noted, however, that a normal electroencephalogram can be present in clinical epilepsy, though this is the exception rather than the rule.

To the insurance physician, this advance in diagnostic accuracy, can be of great help, especially in the underwriting of cases where epilepsy may be denied by an applicant, yet is suspected from the medical history or other records. The chances are approximately four out of five that if epilepsy be

truly present it can be recorded diagnostically by means of this simple procedure, in which there is no more pain, hazard, or expense, than an electrocardiogram. This procedure can likewise be useful to the claims department in evaluating the veracity of a disability claim for epilepsy. Such patients notoriously show little or nothing on neurological examination, so one is dependent on an electroencephalogram for corroboration of the claimant's history when his veracity may be in question. I recall a recent case where a disability claimant, a physician, based his claim on epileptic attacks which had never been observed by other physicians, despite the fact that the patient stated they had been present for from 10 to 15 years. His activities otherwise aroused the suspicions of the company concerned and I was asked to examine him and render an opinion as to his disability. As expected, his neurological examination was completely within normal limits, and his attitude and responses to questions during the examination were such as to likewise question his truthfulness. A completely normal electroencephalogram was most helpful in clinching the diagnosis of malingering and, despite court proceedings, closing of the claim. I greatly doubt whether this case could have been terminated justly without the evidence of a normal "brain wave" recording.

The electroencephalogram can likewise be of great diagnostic help in the epilepsies following head injury, brain tumor or hemorrhage. Here the affected areas in the cerebral cortex can be more accurately localized, a procedure of great help to the neurosurgeon in his subsequent attempt to remove such tumor or posttraumatic scarring.

Therapeutically, new drugs have been discovered which greatly add to our effectiveness in controlling the epileptic seizures. No longer is it necessary to rely on the bromides, with their tendency to toxic accumulation, or phenobarbital and its associated drowsiness. Sodium diphenylhydantoin ("dilantin"), administered in capsules, 3 to 6 times daily, has been found to be the most helpful drug to control grand mal seizures, and fortunately causes no drowsiness as does phenobartital or bromides. The vast majority of cases tolerate

the drug very well but in a small percentage of cases toxic symptoms may become manifest in the form of dizziness, skin rashes, fever or blood dyscrasias. In the petit mal form, trimethadione ("tridione"), similarly administered orally, has vastly improved our ability to control these episodes, hitherto only slightly or not at all helped by phenobarbital or the bromides. This is especially true in the petit mal attacks of children or teen-agers, among whom I have seen patients with as many as 20 or 30 petit mal attacks daily. They completely lose these "blackout" episodes by the use of 3 or 4 trimethadione ("tridione") capsules daily. One word of caution, however, as to the use of this drug. With long continued use it may, though rarely, tend to produce leucopenia which can be serious. Blood counts must, therefore, be regularly obtained at approximately two or three-month intervals while this drug is being administered, and if the white cell count is found to be diminishing to below 4 or 5 thousand cells per c. mm. the use of the drug must be terminated. Fortunately, this is very seldom necessary.

Still more recently, methylphenylethyl hydantoin ("mesantoin") has been added to our list of drugs effective in controlling epilepsy. Though not too thoroughly tested as yet, it has been found helpful in a certain number of difficult cases not adequately controlled with sodium diphenylhydantoin ("dilantin") or phenobarbital. It is only the exceptional case, however, that requires its use.

Because of the above advances in diagnosis and therapy, many more epileptics are able to lead normally adequate and productive lives than hitherto has been the case. It is only the exceptional epileptic these days who has to leave school or employment because of his seizures or mental deterioration. In reference to the latter, it might be noted that such mental deterioration never occurs if the major or grand mal seizures are adequately controlled, as fortunately they can be in most cases today. Petit mal attacks, though a troublesome nuisance to the patient, do not lead to this more serious sequel and even they, thanks to trimethadione ("tridione"), are more effectively handled by the physician.

Can the epileptic, therefore, be safely insured today? In my opinion he can be, with certain qualifications. What should we desire in such an applicant? He should be an intelligent, co-operative individual, who is under the care of a capable physician, who takes his medication regularly, and adheres to the general regime outlined for him, especially abstention from alcohol, avoiding constipation, etc. Irresponsible individuals, the type that "knows all the answers" and is not likely to follow an epileptic regime, are unsatisfactory patients in practice and poor underwriting risks. Fortunately, they are the exception and not the rule. If, while under such observation and treatment, the applicant has had his attacks either completely eliminated, or reduced in frequency to 1 or 2 yearly, if he has been able to carry on adequately in his business or professional endeavors, and if his habits as to alcohol are abstemious, I believe he can be safely insured at some slight to moderate substandard rating. With time and observation, such a rating can gradually be reduced as his favorable course is maintained over a longer number of years. From personal experience I have seen many such cases live out their lives to normal expectancy range or longer, for as some wise physician has said in the past, the best way to live a long life is to acquire some minor ailment and take intelligent care of it.

DR. SIMMONS—Thank you, Dr. Denker. The discussion of epilepsy by Dr. Denker is now open for questions and observations from the floor.

DR. JOSEPH W. JOHNSON—I want to say first how much I appreciate hearing a thought about liberalization in the underwriting of epileptics. The interesting thing is that while the refinements of diagnosis have gone forward rapidly with the use of the electroencephalogram, our refinements of therapy have gone forward just about as rapidly. In the evaluation of any convulsive disorder, epilepsy is but part of the pattern of convulsive disorders, and the encephalogram can bring that out and define and outline a plan of treatment which offers some prognostic usefulness.

The interesting thing about the anti-convulsant drugs, and I think it is important from an underwriting point of view, is that these drugs while effective are potentially dangerous to the bone marrow. With trimethadione ("tridione") particularly, agranulocytosis may occur—rarely in grand mal and much more rarely, of course, with the other anti-convulsant drugs. Methylphenylethyl hydantoin ("mesantoin") is often even more effective in certain cases of grand mal. But the interesting thing about these persons, from my own clinical experience, is that they have an opportunity for survival which is increasing in terms of the effectiveness of the anti-convulsant drugs.

As you know, the electroencephalogram is not generally available. Its use requires a remarkable amount of skill and experience, and there are some individuals—not only the 20 per cent Dr. Denker mentioned—who will not show a characteristic pattern. However, there are characteristic patterns found in the subclinical or preclinical epileptics. In other words, these patients have abnormal waves but do not manifest their disease clinically. Actually, what we are doing, it seems to me, is selecting by aberrant brain waves potential convulsants, as well as more accurately defining the real convulsant. Drugs are becoming increasingly useful in the control of it, because they are increasingly effective and because we do not have mental deterioration to the degree that was formerly apparent with bromides and phenobarbital.

I think that the large companies are in a position to study this subject. Their data will allow us to be much more liberal in the underwriting of any convulsant disorder. I enjoyed Dr. Denker's discussion very much.

DR. LAWRENCE L. MCLELLAN—Dr. Denker, will a patient under anti-convulsant treatment have a normal encephalogram?

DR. DENKER—Yes. I should not say that dogmatically, but when he is under treatment with these medications, then they can suppress the pathological wave. As a rule, they are taken without the treatment.

I would like to make a comment, too, on the remarks of the previous speaker. Although it is true that trimethadione ("tridione") is toxic, sodim diphenylhydantoin ("dilantin") being much less so, in actual clinical experience I have been surprised at how seldom a toxic reaction is seen. A reaction is very much the exception and not the rule. However, there have been two or three fatalities reported from agranulocytosis after use of these two drugs, which I did not mention because of time. Methylphenylethyl hydantoin ("mesantoin"), of course, is used much less than the other drugs. I remember talking with Dr. Resnikoff, the hematologist of Cornell University, about this and remarked that we had used sodium diphenylhydantoin ("dilantin") for ten or fifteen years and trimethadione ("tridione") for four or five years, and I have never seen a toxic reaction. In a place like Bellevue Hospital where many epileptics are treated, I have never seen a toxic reaction. I asked him whether or not he had ever seen one, from the hematological standpoint. He said he never had, but that it must be borne in mind that such things can occur.

Dr. Charles A. Peters—If an encephalogram is normal when using sodium diphenylhydantoin ("dilantin"), it is not much of a help.

Dr. Denker—Well, the drug can be stopped for a day or two before taking the reading. It is of more help from the diagnostic standpoint in questionable cases. It is really not of much help when the attacks are obvious. I do not think it would be of very much help in such cases, but I think it can be of help in borderline cases and in cases where a man is suspected of not telling the truth, either pro or con, from the disability or the underwriting angle.

Dr. Karl W. Anderson—Is it not true that the electroencephalogram is of more value in locating new growths and tumors of the brain than it is in epilepsy? That is the most important piece of work that it has been doing.

Dr. Denker—Well, I was discussing only epilepsy, but there is no question of the value of electroencephalography in other brain lesions, too. Of course, one of the most useful

fields is the localization of brain tumors, various types of obscure sclerosis of the brain, hemorrhagic areas, and so on. There is no question about that.

DR. ANDERSON—Well, this is my point. Epilepsy is merely a minor one of the convulsive disorders, is it not?

DR. DENKER—No, I would not say that. I think from the standpoint of ordinary practice, it is used far more frequently in epilepsy than in brain tumors, and that may be due just to the relative frequency of the two conditions. The ratio is about ten or more epileptic patients to every patient with a brain tumor.

DR. ANDERSON—We have an electroencephalograph in the University of Minnesota and we feel just the opposite. We very rarely use it for epilepsy but use it more for brain tumors.

DR. DENKER—It may be due, again, to the mode of practice and whether most doctors treating epilepsy are taking electroencephalograms on them. But I think the relative frequency between the two conditions is very much weighted in favor of epilepsy.

DR. SIMMONS—Thank you very much, Dr. Denker. I am sorry to interrupt the discussion, but we must go on.

Our next speaker, Dr. James P. Donelan, Medical Director of the Guarantee Mutual Life Insurance Company, will discuss "Orthopedic Impairments."

DR. JAMES P. DONELAN—Frequently the medical underwriter is called upon to appraise risks with orthopedic defects, either congenital or acquired. A rather substantial number of these defects, especially in the juvenile group, may require single or multiple corrective procedures, and even though such impairments have not been investigated by the Joint Committee on Medical Impairments, it should be a comforting thought to the underwriter that the vast majority represent no serious threat to life expectancy.

An orthopedic impairment may be defined as a handicap which deprives the afflicted person of the natural use of some portion of his musculoskeletal system. The term "skeletal" has reference to the tissues of the bones, joints and neuro-

muscular mechanism concerned with the function of the tissues (1). In detail it refers to lost, crippled, paralyzed or deformed individual members of the body, or whole parts of the body. The functional loss could be either total or partial, as a result of amputation, congenital absence of a member, or varying degrees of paralysis due to one of a multitude of causes. The demand for manpower during World War II has brought the problem of the physically handicapped person into sharp focus. The National Health Survey of 1940 indicates that there were an estimated 1,844,600 males in the United States with orthopedic impairments of whom 208,500 were incapacitated. In addition, a total of 759,000 females with such impairments was estimated, of whom 132,500 were incapacitated and 626,500 had non-disabling impairments. The average period of disability caused by an orthopedic impairment during the year preceding the survey was above eleven months for all ages (2). The majority of non-disabling and a substantial number of major defects do not affect life expectancy. I will endeavor to briefly summarize the consensus of underwriting practices related to frequently encountered orthopedic impairments.

Residual Paralysis Acute Anterior Poliomyelitis:

Many victims escape paralysis from this common, acute, virus disease. Those patients who develop residual paralysis become problems that we must consider from an underwriting point of view and many can be insured at standard rates; waiver of premium and accident benefits can be included if but little impairment of function exists. Risks with involvement of one leg or one arm can be assumed at standard rates; waiver of premium and accident benefits probably should be granted with a slightly increased premium. More extensive muscular involvement requires careful individual handling. There are relatively few such cases presented for consideration and the opinion of an orthopedist regarding the extent of paralysis, and deformity or functional loss would permit

a more exacting appraisal than the routine medical examination. The expense incurred would be far outweighed by accurate information gained.

Amputations:

Trauma, congenital conditions, infection, malignant growth and vascular disease make up the majority of causes for amputation or disarticulation of an extremity. When appraising an amputee for life insurance, our first concern must be the cause; our secondary concern is to what extent the loss or partial loss of an extremity affects physical activity and vocational ability. We must also make certain that sufficient time has elapsed since the amputation was performed to assure rehabilitation. The Medical Impairment Study of 1929 indicates that in a small group of Standard lives with one leg amputated above the knee, the ratio of actual to expected deaths was only about 10 per cent in excess of that which could be considered normal mortality. In a somewhat smaller group of substandard lives the ratio was found to be nearly 40 per cent in excess of that normally expected.

Company ratings for these two groups as a whole averaged 140 per cent. No cause of death was significant, and there were only four deaths from accident against seven expected. Since this study, many advances have been made in the care of persons with amputations. World War II taught the value of bringing patients together under the direction of specialized surgeons in so-called amputation centers. These surgeons had the responsibility not only for organizing the whole functioning of the center but for the complete care of the patient and all of his problems. Such good planning and organization are adaptable to use in civilian life in a limited way. Experiences gained in care and rehabilitation of amputees at such centers will undoubtedly reflect in the care of civilian amputees and will help lessen the economic and accidental hazards that have chiefly concerned us in underwriting such risks.

Amputations or disarticulations because of trauma or congenital problems, are accepted in my company with the fol-

lowing ratings subject to medical examination:

Loss	L. R.	W. P.	D. I.
One Arm	Standard	+50%	+50%
Both Arms	$2.50 per M	No	No
Leg or thigh below upper ⅓ with well fitting prosthesis	Standard	+50%	+50%
Same as above, crutch used; or both legs below knees with prostheses	$2.50 per M	No	No
Both thighs middle or lower ⅓—usable prostheses	$5.00 per M	No	No
One arm and one leg; or one leg above mid ⅓	$5.00 per M	No	No

Amputation performed because of extensive infection or malignant growth must be underwritten with caution, particularly so during the first five years following surgery because of the possibility of a recurrence. The attending physician's statement should be reviewed, and careful examination of the amputation site is imperative. Applicants with amputations performed because of vascular disease or diabetic gangrene are uninsurable.

Low Back Pain:

Many reviews and summaries on the problem of pain in the lower part of the back appear in the literature. In most of these, sciatica is discussed as a secondary feature. Postural strain, round back and epiphysitis seen among adolescents, spondylolisthesis (3) and spondylolysis, the etiology of which is uncertain, myositis and fibrositis, occurring after periods of rest, so stubbornly resistant to treatment (4), are but a few of the causes of low back pain. The rôle of the sacro-iliac joints has assumed smaller proportions in the past decade while the lumbo-sacral joint and hypertrophied ligamentum flavum have increased in importance as etiological factors. Longevity is but little affected; recurrent periods of disability are common.

New light was shed on the cause of low back pain in 1934 when the syndrome of herniating, or ruptured intervertebral disk was described (5). Since then thousands of patients have been operated on and numerous reports have been made in the literature. In earlier days, caution led to the visualization of the herniating disk by myelography, following injection of a contrast medium. Time and experience have led to the conclusion that chronicity, unilaterality and typical radiation of sciatic pain means herniating disk until proven otherwise. The diagnosis is known to be accurate in 90 per cent to 95 per cent of cases (6). The syndrome of herniating intervertebral disk is predominately that of pain in a nerve root as the protruding portion of the disk compresses a nerve root against the ligamentum flavum adjacent to it. Pathologic disks usually occur below the fourth or fifth lumbar vertebra. Once a disk has been diagnosed, repeated cycles of pain can be anticipated. The results of surgical treatment reported by the neurological surgeons are highly satisfactory. Disability is reduced from almost total during acute exacerbations to 5 to 10 per cent after surgery in most cases. One operation is usually pain relieving; occasionally a second operation for a missed disk or spinal fusion will be necessary. Mortality incident to disk surgery in good hands is negligible.

All risks with a history of pathological lumbar disk within a period of five years should be examined. If one or more cycles of pain have occurred, surgical removal must be considered in our underwriting approach to such cases. Unoperated cases can probably be assumed with an extra mortality of 50 per cent. We cannot grant double indemnity or waiver of premium to the unoperated group. Operated cases should be investigated through the attending neurosurgeon and if favorable results are anticipated, these cases may be accepted for life insurance at standard rates six months after recovery. Accidental death benefits may also be included after a six month recovery period. Waiver of premium should be granted cautiously within two years after surgical removal.

Scoliosis:

Scoliosis may be defined as a rotary lateral curvature of the spine (7). This may be either functional or structural. Functional curvatures are usually due to some non-spinal involvement such as faulty posture, the shortening of one lower extremity, etc. Such problems are correctible when the non-spinal defect is discovered and relieved. Risks with such defects are underwritten on the basis of the non-spinal defect.

Structural scolioses are due to involvement of normal bone, nerve and muscle balance. Individuals so affected are unable to maintain the spine in normal alignment (7), and varying degrees of spinal and thoracic deformity may exist. It can be assumed that approximately 20 per cent of the persons with spinal curvatures can give adequate cause for the deformity, 80 per cent of them cannot (8). These, of necessity, are classified as idiopathic curvatures. Known causes listed are residual paralysis of poliomyelitis, congenital anomalies such as hemi-vertebra, Sprengel's deformity, neurologic diseases such as Friedreich's ataxia, and muscular dystrophy. About 75 per cent of spinal curvatures will be found in young females, dating the onset at ages 10 to 12 years. They usually seek orthopedic care in the early teens. Practically all idiopathic curvatures stop progressing at the cessation of the growth period. Corrective exercises aid in establishing compensatory curves; mechanical supports and braces are used with limited success in arresting structural curves but spinal fusion is apparently the only method of treatment that will prevent increase and maintain correction of advancing spinal curvature.

Insurance can be granted at standard rates to individuals beyond the age of 16 years who have but slight structural curvatures. Waiver of premium and double indemnity can be included. A risk with a moderate to extreme curvature should be examined. X-ray studies including anteroposterior and lateral views are advisable to determine the degree of deformity of the spine and thoracic cage. If the thoracic viscera are displaced, the risk should be rated substantially, as individuals so impaired will show an increased mortality from organic disease of the heart and lungs.

In conclusion may I repeat that significant progress has been made during the past few years in the field of orthopedic surgery. Antibiotic and chemotherapeutic agents have hastended healing and decreased the number of complications from surgical and non-surgical problems. However, we should proceed cautiously in revising our present underwriting practices as they refer to orthopedic impairments, until sufficient time has elasped to definitely establish that healing of relatively short duration results in permanent cure.

DR. SIMMONS—Are there any questions that you would like to direct to Dr. Donelan? I assume, then, that you are all in agreement with him, and we will proceed to the next part of our forum.

I would now like to introduce to you Dr. Robert L. Weaver, Associate Medical Director of The Penn Mutual Life Insurance Company, who will discuss "Osteomyelitis."

DR. ROBERT L. WEAVER—I am sure that most of us are cognizant of the etiologic, bacteriologic, and pathologic features of osteomyelitis, but perhaps a few will benefit, as I have, from a quick review as a preface to this short discussion.

Briefly, osteomyelitis is an infection of bone, derived by direct implantation of bacteria as the result of local trauma, compound fracture, gunshot wound, or metastasis incident to the bacteremic or septicemic phase of an acute infection. The greatest incidence occurs in childhood, ages two to ten. Adult infections, while they may be primary, are usually recurrences of childhood infections. Males are affected twice as frequently as females. The long bones, especially the tibia, femur and humerus are the common sites. Key (9) states that under two years of age the staphylococcus aureus is the offending organism in more than 90 per cent of cases; over age two in about 50 per cent of cases. Next in frequency are the staphylococcus albus, hemolytic staphylococcus, streptococcus, pneumococcus, micrococcus and proteus organism.

As a result of thrombosis of blood vessels, bone necrosis proceeds with suppuration, and if not controlled, sequestration; concomitantly, osteoblastic repair and formation of new

bone, or due to ineffectual attempts at healing, a chronic phase ensues. This is manifested by cavities of varying size containing infected granulation tissue, necrotic bone, purulent matter which may or may not yield an organism on culture, sinus tracts, and scarred fibrous tissue. Reparative processes are manifested by periosteal and endosteal new bone formation with resultant obliteration of the Haversian canals, medullary canal, and diminished to absent blood supply. The periosteal covering is thickened and fibrous.

It is important to fix this picture in one's mind to understand why the sulfonamides, penicillin, and other antibiotics are ineffectual by themselves in the chronic phase. These substances are effective only in *adequate concentration* against *susceptible* organisms, in the presence of an *adequate* blood supply. In the chronic phase we are dealing with a bone abscess shielded by avascular, dense, necrotic bone, surrounded by purulent matter and covered by poorly nourished, thickened periosteum. Truly a serious problem and not to be solved by routinely administered antibiotics.

Those of us who saw osteomyelitis before the advent of adequate surgery aided by penicillin, will never forget the results of repeated surgical drainage, staphylococcus toxoid, and in desperation, treatment with maggots; nor the crippled, stiffened joints, atrophied muscles, pathological fractures, chronic sepsis, anemia, recurrences, and amyloid disease as end results.

In acute hematogenous osteomyelitis the outlook seems particularly bright, and penicillin in adequate dosage seems effective without operation, if given early enough. In fact, Key advocates prompt and early chemotherapy when the condition is only suspected and before bacteriological identification has been made; this is based on the fact that vascularity still obtains, so that bacteria are exposed to tissue fluids in contact with an active blood stream carrying an optimum concentration of penicillin. Extension, dissemination of the disease, and thrombophlebitis are obviated, surgery becomes elective if at all necessary, when fluid balance and anemia are under control.

Wilflingseder (10) reports a series of 18 children with acute hematogenous osteomyelitis treated by intramuscular administration of penicillin; one million to two million units proved to be sufficient when treatment was initiated in the first few days of the disease. In 17, complete recovery occurred in the absence of sequestration and formation of fistula. In one case involvement of the joint and sequestration occurred because of too small a dosage. Fontaine and Grosse (11) report a series of 12 patients, 7 boys and 5 girls between the ages of 4 and 16, with acute osteomyelitis of the long bones treated by penicillin. Five patients in whom penicillin was started immediately at the onset of the disease, showed prompt recovery without abscess or bone involvement. Follow-up continued for five to twelve months. Three suffered recurrences in three weeks to two months; the balance, because of delayed treatment or grave condition at onset, were saved, but a chronic course with abscess and sequestra could not be avoided. Fontaine and Grosse feel that penicillin is not the treatment of choice. Their dosage, five hundred thousand to one million units every three hours intramuscularly, seems small in comparison with other series reported and may have been inadequate.

Chronic osteomyelitis, however, definitely remains a problem despite penicillin, because of the pathology mentioned previously. Here we are dealing with a bone abscess devoid of blood supply and, therefore, unaffected by penicillin alone without surgical intervention.

Orr's (12) technique, which has prevailed since 1927, was good considering our knowledge then and the armamentarium available to us. However, despite careful attention to principles, drainage often persisted many months, and secondary reconstruction methods were delayed. Split skin grafts were used, as well as techniques described in 1902, 1922 and 1926, but failed because of pockets forming beneath.

Newer surgical principles, developed as a result of World War II, and penicillin have definitely changed the picture today. These include the knowledge that cancellous bone has a higher resistance to infection than cortical bone, and

the use of cancellous bone chips to fill in bony defects, described by Coleman (13). Coleman also believes in primary closure in the presence of infection, despite Dickson's (1944) teachings. Prigge (14) described the use of muscle transplants and iliac graft; Stark (15), the use of pedicled muscle flaps in the reconstruction of chronic osteomyelitis resulting from compound fractures. So that today, although techniques and teachings differ somewhat, particularly as to the time of closure in the presence of infection, the general principles are pretty well uniform. These consist of preoperative penicillin while anemia and fluid balance are corrected. Adequate excision of the entire osteomyelitic cavity beyond just sequestrectomy down to healthy granulating bone, with saucerization to eliminate overhanging edges, the use of cancellous bone chips to fill in bony defects and primary or secondary closure, supplemented by split skin grafts, or pedicle muscle flaps, and postoperative penicillin to protect the reconstruction measures until healing has obtained. It will still take some time to determine the end result, but the trend and the outlook are certainly very bright.

A recent report by the Mayo Clinic (16), in a series of 68 cases treated during 1944, 1945 and early 1946, indicated that the average hospital stay was twenty to twenty-four days, depending on whether there was primary closure or drainage. The average dose of penicillin was one hundred thousand to one hundred forty thousand units per day for fourteen to fifteen days. Complete healing obtained in 67 per cent of the drainage cases and 88 per cent of the primary closure cases.

McClintock (17) reports on 150 cases, 148 of which had had infection existing three months or longer. After penicillin, excision, saucerization and plaster dressing, he carried out skin grafting on the sixth to tenth day. Sixty-six of his cases healed in twenty-five days; 69 in fifty days. X-ray appearance of the affected bone is described as almost normal as a result.

In view of all these changes, it is pertinent for each of us to individually review our ratings for acute and chronic osteomyelitis. Consequently, inquiries were sent to twenty

companies and replies were received from nineteen, which, together with my own, make twenty. It is very difficult to summarize the various methods of treatment of this impairment by companies, because of different classifications. However, an attempt has been made to do so in the following tables.

It would appear that a single attack involving a single bone is declined by most companies for a period of one year. The second year the average rating is plus 75 per cent extra to plus 50 per cent extra; the third year, plus 50 per cent to plus 25 per cent extra. After the third year the average rating seems to be plus 25 per cent extra to zero. Most companies will grant Waiver of Premium, if there is no substandard rating.

Recurrent attacks are declined by most companies during the first year; many for two years. The average rating of those who accept in the second year is plus 75 to plus 50 per cent extra, carried through the fifth year; from the sixth to the tenth year, plus 50 to plus 30 per cent extra; tenth year and later, plus 25 per cent extra to zero.

Those companies that do accept either of these impairments within the first year seem to rate a single attack, single bone, plus 50 to plus 25 per cent extra; recurrent attacks, plus 100 to plus 50 per cent extra.

Four companies have recently made modifications in their rating because of the results obtained by chemotherapy and up-to-date surgical techniques.

Five companies will accept even though a draining sinus is present involving one bone, depending upon the location and the amenability to amputation, rating plus 50 per cent extra.

Eight companies that accept lesions in multiple areas, add plus 50 per cent to plus 25 per cent to their ratings.

In general, most companies temper their ratings for osteomyelitis of the small bones of the extremities, or the jaw secondary to an abscessed tooth, or where the process is amenable to amputation.

Single Attack

Company	0–6 mos.	6–12 mos.	2nd year	3rd year	4th year	5th year	6th year
#1	Decline	Decline	+75–+50	+40–+25	+40–+25	+40–+25	+20–0 W.P.
#2	Decline	Decline	Cautious and tend to standard if x-ray negative on account of penicillin				
#3	Decline	+50	+50	0			
#4	Decline	Decline	+75–+50	+75–+50	+25	+25	+15
#5	Decline	Decline	+60	+60	+30	+30	0
#6	Decline	Decline	Regard favorably				
#7	Decline	Decline	+75–+50	+50–+25	+50–+25	0	
#8	Decline	Decline	Decline	Decline	Decline	Decline	(Individualized, however)
#9	+50–+25	+50–+25	+25–0	+25–0	+25–0	+25–0	0
#10	Decline	Decline	+50–+25	+50–+25	+50–+25	+50–+25	0
#11	Decline	Decline	+100–+50	Later action dependent on amount of damage and discharge present			
#12	+50–+30	+50–+30	+50–+30	+30–0			
#13	+60–+30	+30–0	+0				
#14	+50	+50	+50–0	+0			
#15	+25	+25	+25	+25	+25	+25	0
#16	+25	+25	+25	+25–0	+25–0	+25–0	0
#17	+25	+25	+25	+25–0			
#18	+25	+25	+25	+25–0			
#19	Decline	Decline	+75–+25	+75–+25	+75–+25	+75–+25	+25–0
#20	Decline	Decline	+75–+50	+40–+25	+40–+25	+40–+25	+25–0

Multiple Attacks

Company	0–6 mos.	6–12 mos.	2nd year	3rd year	4th year	5th year	6–10th year	10th year & later
#1	Decline	Decline	Decline	+100 – +50	+100 – +50	+100 – +50	+40 – +15	+15 – 0
#2	No data							
#3	Same as for single attack							
#4	Decline	Decline	Decline	+75	+75	+75	+50	+25
#5	Decline	Decline	+80	+80	+80	+60	+60 – +30	+30 – 0
#6	No data							
#7	D – +100	D – +100	D – +100	D – +100	D – +100	D – +100	+75 – 0	
#8	No data							
#9	+75 – +50	+75 – +50	+75 – +50	+50	+50	+50	+25 – 0	+25 – 0
#10	Decline	Decline	Decline	Decline	+75 – +25			
#11	Decline	Decline	D – +100					
#12	+100 – +50	+100 – +50	+75 – +30	+50 – +30	+30 – 0			
#13	Decline	Decline	D – +100	D – +60	D – +60	D – +60	+60 – 0	
#14	+50	+50	+50	+50	+50	+50 – +25	+50 – +25	
#15	D – +50	D – +50	+50	+50	+50	+50	+25 – 0	
#16	+50 – D	+50	+50	+25 – 0				
#17	+50 – D	+50	+50	+25 – 0				
#18	+50 – D	+50	+50	+25 – 0				
#19	No data							
#20	No data							

It seems to me that, although we can propose certain ratings in view of recent developments, many of these cases will have to be individualized, depending on the bones involved, the quality of medical and surgical treatment, and the availability of x-rays to clarify the clinical history. While there may be some companies willing to accept cases wherein multiple bones are affected, I do not feel that the experience will be good. Certainly, there is no question but that osteomyelitis of the skull and bones which are not readily accessible to surgical intervention should carry heavy ratings, and that we can temper the rating where small bones of the extremities are involved, or processes amenable to amputation.

I have therefore constructed the following table of ratings for osteomyelitis, not tuberculous, as a suggested schedule based on the above discussion. These ratings represent merely my own personal point of view.

Osteomyelitis — Not Tuberculous

Proposed Rating

	Rating	Waiver of Premium
Single attack		
0 to 3 months	Decline	No
3 months to 1 year	+50 to +30	No
2nd year and later	+25 to zero	Standard
Recurrent attacks or multiple bones affected		
0 to 1 year	Decline	No
1 to 2 years	+75 to +50	No
2 to 5 years	+50 to +25	No
5 to 10 years	+25 to zero	P. R. N.

Quality of surgical treatment should be given consideration.

Rating modified or increased depending on x-ray findings, number of bones affected, and whether or not amenable to amputation.

Osteomyelitis of jaw secondary to abscessed tooth, and osteomyelitis of small bones of extremities may be treated more liberally.

DR. SIMMONS—Thank you, Dr. Weaver. Dr. Weaver's paper is now open for discussion. Are there any questions from the floor? I assume they are in agreement with you, Dr. Weaver.

Dr. J. Gilbert Falconer, Medical Referee of the North American Life Assurance Company, will now discuss "Chronic Cholecystitis."

DR. J. GILBERT FALCONER—The mortality studies published in regard to chronic cholecystitis are those of the Medical Impairment Study of 1929, and the paper read before this Association by Drs. Dublin, Jimenis and Marks (18), in 1934. I can find no more recent detailed study in the literature of insurance or clinical medicine. The difficulties which we meet in the underwriting of chronic cholecystitis seem to me to arise from two main causes. The first is the matter of the practical application of the above mentioned studies to underwriting. The second is the matter of inaccurate diagnosis by the attending physician. In spite of improved radiologic and other techniques, there is still a great tendency to label any form of indigestion associated with belching of gas as chronic cholecystitis.

Chronic cholecystitis without stone is not commonly the cause of gastro-intestinal complaints, although it is not an uncommon finding at post mortem examination. Occasionally an inflammatory scarring of the common duct or peri-cholecystitis may cause symptoms, but current opinion would suggest that patients who have severe enough symptoms to warrant operation will have gall stones in 80 per cent of cases. It is probable that the large majority of cases which are labelled chronic non-calculus cholecystitis are in fact functional digestive disorders with pylorospasm as the immediate cause of distress. One must, of course, recognize that some of these patients while not suffering from gallbladder disease are suffering from some other serious condition which has not been discovered, namely peptic ulcer, pancreatic disease, small bowel disease, or disturbances of the large bowel, particularly in the region of the cecum or hepatic flexure. In Dr. Dublin's paper the whole group of gallbladder disease with a history of

medical treatment showed a mortality of 115.1. A subdivision of these figures showed a mortality of 136.7 for cases classed as inflammation of the gallbladder. However, this diagnosis was based largely on the applicant's statement, and it is stated earlier in that paper that it was not customary to refer to the attending physician for details of the illness. It is probable, therefore, that those cases labeled inflammation contained a great many different conditions which under more recent methods of underwriting would have been eliminated from the series. It is very difficult to believe that their group shows the true mortality of uncomplicated chronic non-calculus cholecystitis. In underwriting these risks it would seem safe to overlook the cases which appear to be actually mild functional indigestion and to allow for an extra mortality of 40 or 50 per cent in those which are more severe or have frequent recurrences, or where there seems to be some possibility of the presence of other organic disease.

Chronic calculus cholecystitis is a very definite hazard to longevity. In this group, I am presuming that the diagnosis has been established by one or more clear-cut attacks of gallbladder colic or by radiologic investigation. The risk of operation on an uncomplicated case is, of course, not great but there is always the possibility that the surgeon will find a stricture or stones in the common duct which make the operation more extensive and undoubtedly increase the risk of post-operative complications, immediate and remote. Our present method of rating these applicants when they have had only one attack seems to be reasonable. The question arises, however, when the applicant has had two or more attacks, of the advisability of insuring him at any rating within one year of his last attack. The extra hazard will, of course, arise from the fact that the greater the number of true attacks of gall stone colic, the more certain it is to require operative interference at a later date. There is also the fact that the cases of acute cholecystitis with all its attendant complications arise largely from this group.

I now come to another subdivision of chronic cholecystitis which I believe warrants more attention than we have paid

to it in the past. This is acute cholecystitis. I term it a subdivision of chronic cholecystitis because it is almost invariably engrafted upon previous gallbladder disease. Many of these patients will temporarily recover without operation but the condition is very likely to return at a later date and surgery will be necessary. The many complications of this condition which may arise pre-operatively or post-operatively are well known. It is also to be noted that in the Medical Impairment Study of 1929, and in Dr. Dublin's paper, the only definitely increased mortality appeared in the cases which had had surgical drainage of the gallbladder. A case of acute cholecystitis treated medically or with surgical drainage of the gallbladder comes within the scope of our discussion because in neither case has cholecystectomy been performed. We all know that acute cholecystitis is a serious disease and it seems to me that if cholecystectomy has not been performed this group is likely to show a very unfavorable mortality. The Medical Impairment Ratings suggests that "infected gallbladder, no stones, surgical drainage" and "infected gallbladder, no operation" be rated the same as "gall stones, no operation". I find, also, that some companies are rating cases of infected gallbladder, where presumably the physician has been unable to demonstrate the prescence of stones and there has been no operation, at even less than one attack of so-called gall stone colic. If cases of acute cholecystitis are included in this group, I am sure that the mortality will ultimately turn out to be high. It is possible, however, that this reduced rating is being reserved for the ordinary doubtful case of chronic cholecystitis.

I would suggest for your consideration that we add to our system of classification another group under the heading of gallbladder disease and label it "acute cholecystitis without removal of gallbladder". This group would be relatively small, but would cover those cases which have had an illness characterized by marked pain and tenderness in the right upper quadrant usually associated with fever and vomiting and lasting for at least several days and possibly for several weeks. I would suggest that these not be accepted at all

until the second year after one atack, and then the rating should be probably plus 100, and that if they have more than one attack they not be accepted until the third year after the last attack and the rating should then be at least plus 150.

Your further comments and suggestions will be gratefully received.

DR. SIMMONS—Is everyone in agreement with Dr. Falconer on this important subject? Are there any questions or discussion?

DR. THOMAS H. DICKSON—I would be very glad if Dr. Falconer would comment briefly on these cases in which we have a history of digestive difficulty, perhaps over some time, and the only thing we get from the attending physician is a report of "non-functioning gallbladder."

DR. FALCONER—I think that probably those cases have been serious enough to suggest that the attending physician carry out radiologic investigation. If there was no filling at all of the gallbladder on the second attempt, one would have to conclude that there was likely gallbladder disease of one sort or another. Of course, quite possibly, there may be non-opaque stones, and I should think that group would come under the classification that I suggested might carry a 40 or 50 per cent extra mortality. On the other hand, the common radiologic report of poor filling, or rather small but evenly outlined gallbladder, I think does not amount to much, and I would suggest that such cases are functional digestive disorders, or some other disease.

DR. ANDERSON—The cases that bother me are those with reports from around the midwest—the Mayo Clinic and other clinics of that character—where many people go now for purely routine examinations. They report, "solid stones by x-ray," or just say, "a gall stone by x-ray." These people, without symptoms of anything, go just for a routine examination. What do you do in those cases?

DR. FALCONER—Well, I think when a stone is found accidentally like that, especially one large stone, and there is reason to believe that they are truly symptom free, one would

have to consider them definitely substandard. Probably they are no worse than those with the rating for one attack of gall stone colic. It is much less common, I think, for the small multiple-stone cases to remain symptom free than those with the single large round stone.

DR. SIMMONS—Thank you, Dr. Falconer. Our final speaker on the forum is Dr. Berthold T. D. Schwarz, Vice President and Medical Director of the Bankers National Life Insurance Company, who will speak on "Asthma."

DR. BERTHOLD T. D. SCHWARZ—Our president, Dr. Robinson, requested me to report at this meeting a brief summary of present day thinking and underwriting practices with respect to asthmatics. My report is based on replies from 116 of the 234 member companies comprising this Association, accounting for 87 per cent of paid-for new insurance in 1947. The majority of medical directors replying expressed the need for greater uniformity in definition of terms, clearer classification, and a better conception of modern diagnosis and treatment. They felt that a more liberal view was justified. A smaller number were satisfied with present practices, while only a few thought this impairment should be appraised more severely.

Examination of the various underwriting rules confirmed the need for modification of our practices. These generally adhere to the Medical Impairment Studies, although they are based on four investigations between the years of 1895 and 1936. They include a study (19) by the Metropolitan Life Insurance Company. Our 1940 study (20) presented a critical analysis of these in detail.

It should be understood that the field of allergy, including its important etiologic rôle in asthma, is only a comparatively recent development in medicine, dating back to 1906. It is only within the past fifteen to thirty years that the pathogenesis and clinical application of this new field have come to occupy their proper place. Our Medical Impairment Studies and the underwriting rules based upon them do not reflect these advances in medical practice. Life insurance studies of asthma are all based upon the symptom and sign of

wheezy respiration of heterogeneous origin, whereas in clinical studies the word asthma refers only to bronchial asthma, a distinct homogeneous clinical entity. This explains the prognostic difference between the life insurance and the clinical conceptions of asthma. Asthma may be referred to as a symptom complex rather than a disease. Hence, for all life insurance investigations confusion would be avoided if the pleural form of the word is employed. It is obvious that until properly investigated and classified the etiology of all asthmas remains obscure. These are called *idiopathic asthmas,* and the term should cover the symptom of paroxysmal dyspnea of *unknown etiology.* Investigation of the cause and clinical features permits further classification into those of *known* etiology, of which there are only two kinds, *primary* and *secondary. Primary asthma is bronchial asthma,* and there are two kinds of *bronchial asthma, extrinsic* and *intrinsic.* When the unqualified word asthma is used, it should refer only to the complex symptomatology constituting the entity called bronchial asthma. *Secondary asthma* is that of *known etiology* where the symptom of paroxysmal dyspnea is caused by some known primary disease such as bronchiectasis. By adhering to etiologic definitions, all asthmas may be easily segregated and best analyzed and evaluated according to whether the etiology is known or unknown, as follows:

Class I: Asthma—known etiology—A Primary; B Secondary; C Mixed.

Class II: Asthma—unknown or *undetermined* etiology—*Idiopathic* (paroxysmal dyspnea).

In analyzing this etiologic classification, the salient etiologic and clinical factors are discussed below under their respective classified subdivisions. Preceding these various classes and subdivisions are broad recommendations for use in the selection of such risks. Much of the preliminary work in medical selection, such as the investigation for classification, may be more understandingly performed by lay underwriters using etiologic factors. But, because of the wide range of synonyms used in discussing asthmas, many of these risks will present major problems for classification and estimation of longevity.

CLASS I: ASTHMA—KNOWN ETIOLOGY

A *Primary* (Bronchial Asthma)—*MAJORITY ACUTE—STANDARD; CHRONIC—SUBSTANDARD TO RISK NOT ACCEPTABLE.*

Associated with physical signs of respiratory tract highly sensitive to all types of intrinsic and extrinsic stimuli. Causes, various and complex, produce different clinical types, sometimes a confusing mixture of these types. Demonstrable etiology in 80 to 90 per cent of all cases makes prognostic classification easier. This type easily subdivided into two groups—*extrinsic* and *intrinsic.* Those of extrinsic origin constitute 80 per cent, according to Rackemann (21); intrinsic 20 per cent. Bronchial asthma usually benign in acute varieties. Primary asthma often referred to as true, benign, uncomplicated, essential, atopic, allergic, non-allergic (reflex), anaphylactic, idiosyncratic and acute or chronic, depending upon interval between attacks. Attacks may be *seasonal* or *perennial.* Chronic types termed intractable, status asthmaticus. If etiology doubtful, consider under asthma—Unknown Etiology, Class II.

a. *Extrinsic*—due to allergy or idiosyncrasy.

1. Allergy incited by specific proteins, molds, dusts, smuts, rusts, and bacteria. Allergy defined by Urbach as a "condition of altered tissue reaction to substances or stimuli of a physical or chemical nature which in similar amounts provokes no response in the majority of members of the same species." Rubin (22) states nature of trigger mechanism setting off response of tissue not entirely clear. These reactions sometimes called *anaphylactic.* Allergy referred to by some as atopy. Allergens produce demonstrable antigens in the body. These *allergens* and *idiosyncratic substances* commonly present in the atmosphere or environment. Of major etiologic importance, as a change in occupation or environment results in cessation or freedom from attacks. Yield to desensitization therapy. Asthma caused by *pollens* generally of seasonal variety, acute in character, long intervals of freedom. *Occupational dusts* or *food* likely to be *perennial.* Either

variety generally very *amenable* to treatment. Both cause *varying intensity* of attack *depending* on the *sensitivity* of patient and *amount of asthmogenic agent.* Climate or environment actually atmosphere plus or minus asthmogenic substances.

2. *Idiosyncrasy*—reaction to substances of a chemical nature—i.e. aspirin, ipecac, produce no antibodies. Believed to combine with protein-forming complex molecule (hapten).

b. *Intrinsic—cause* originates from *within* the person. These essentially unaffected by changes in *climate, environment,* or *occupation.* May be allergenic or nonallergenic (reflex). *Less amenable* to *symptomatic* and *curative therapy.* Apt to be *perennial* and *chronic* in character. Occur months after history of acute *suppurative* respiratory tract *infections.* Aerosol antibiotics effective in many. Bronchial asthma this type definitely *attributable to* a *specific cause* is a small group. Majority of cases in primary intrinsic category (known etiology) eventually fall into (known) secondary classification. Until the etiology is determined, classed as unknown—idiopathic asthma (23) .

B *Secondary—GENERALLY HIGHLY SUBSTANDARD, OR RISK NOT ACCEPTABLE.*

Secondary asthma also referred to as complicating, spurious, thymic, cardiac, renal, neoplastic, or malignant. Classify and underwrite primary condition, viz. bronchitis, suppurating sinusitis, emphysema, bronchiectasis, myocarditis, enlarged glands, or cicatrices producing bronchial stenosis, and growths. Most of these risks not acceptable and present no selection problem.

C *Mixed—MAJORITY—STANDARD; IF ASSOCIATED WITH INFECTION—SUBSTANDARD.*

May eventually become intrinsic or secondary asthma. Occur under the following divisions:

1. Combination Class I, A and B.

2. Nasal polyps, obstruction and irritation of nerve endings, or infection (so-called bacterial sensitivity), or both. Reflex or bacterial allergy, or both. These most numerous, usually benign.

3. Clinically important group known as "infectious asthma", or "asthmatic bronchitis". Bacterial sensitivity causing allergic bronchial asthma, reflex (non-allergic), secondary asthma, or a combination of these. Often relieved by aerosol penicillin and streptomycin.

4. Focal infection from tonsils, teeth, gallbladder, prostate, etc.

CLASS II: ASTHMA—UNKOWN ETIOLOGY

Idiopathic (Paroxysmal Dyspnea)—*MAJORITY—STANDARD; SOME—SUBSTANDARD TO RISK NOT ACCEPTABLE. CONSIDER ON BASIS OF BROAD CLINICAL TYPE, FUNCTIONAL ABILITY, AND THERAPEUTIC RESPONSE.*

All asthmatics not clearly classifiable in Class I, A, B, or C. This group constitutes main challenge in medical selection. All asthmas have a *common fundamental flaw.* Details differ as to precipitating cause for attacks. Frequently acquire sensitivity to additional causes, probably resulting in a *fundamental* disturbance in normal physiologic mechanisms of the body. These probably endocrine, in turn probably effected by a myriad of imbalances between metabolism of foods, minerals, and vitamins necessary for proper smooth physiologic response. These probably reflex (non-allergic) types. If their obscure etiology reasonably clarified, may be reclassified as probably Class I, A (Primary, mixed extrinsic and intrinsic) (23).

a. Psychosomatic conditions
b. Physical or thermal irritations
c. Obscure atmospheric and meteorologic conditions
d. Biochemical alterations
e. Endocrine imbalance (thyroid disorders, menstrual cycle, and interruption by pregnancy)
f. Vitamin dyscrasias

SUGGESTED METHODS FOR DETERMINATION OF CLASSIFICATION

For proper prognostic classification, asthmatics in Class II should be thoroughly investigated as to the probable etiology.

In a condition so complex in its manifestations, this may be done best by using one or more of the following questionnaire methods. The ones selected should be those most adaptable to the individual case.

1. *History* forms of the type commonly employed by allergists may be suitable, or these may be altered as desired.

2. A chart based on the *broad clinical type,* from the differential diagnosis point of view, is outlined in Table 1.

3. Prognosis may be estimated also by determining the *degree of impaired functional ability* and the *character of response to therapy.* This is outlined in Table 2.

4. Finally, a *special clinical type* of classification is listed in the following outline form, under the headings A Allergic (atopic), B Mixed, and C Infectious (24).

A *Allergic (atopic)—ACUTE TYPES—STANDARD; CHRONIC, ESPECIALLY IN OLDER AGES—GENERALLY RISK NOT ACCEPTABLE.*

General clinical findings, positive personal, family, food and inhalant history. Other allergic manifestations (eczema, hives, migraine, pallor of mucosa and often of uvula, cough near end of attack, sputum glairy, mucoid and frothy, positive cutaneous reaction, asthmatic breathing). Good response to desensitization, avoidance, or medications.

a. *Acute*—Paroxysms relatively short, followed by prolonged periods of practical normal well-being. May have paroxysms of dry cough and slight wheezing. Response to treatment usually prompt; more intense therapy necessary in older ages.

b. *Chronic*—Orthopnea severe lasts weeks, response to treatment fleeting. Intractable asthma, often symptomatic of secondary asthma, may be of malignant type. Chronic cough, dyspnea, strenuous efforts to expectorate followed by transient relief. Often called asthmatic bronchitis, of mixed type, generally complicated with sinusitis, bronchitis, emphysema and bronchiectasis. Status asthmaticus—end result of grave state of respiratory impairment.

Table 1. Classification of asthmas not clearly Class I and those of doubtful insurability, based on broad clinical type, including age at onset, frequency, duration, severity, and time elapsed since last attack, response to therapeutic measures. Check applicable box.

BROAD CLINICAL TYPE DIFFERENTIAL

1. Age at first attack_____
2. Date of last attack_____
3. History of ☐ hay fever ☐ other allergy ☐ family history ☐ none
4. Longest interval (weeks, months, or years) between attacks—specific dates _____

	Positive	Probable	Suspect
5. Seasonal—usually acute paroxysms in pollen season	☐	☐	☐
6. Perennial—year round, with irregular recurring respiratory symptoms (rhinitis, chronic cough, wheezing), rare acute paroxysms	☐	☐	☐
7. Occupational—	☐	☐	☐

8. Probable cause of attacks_____

9. Character and frequency of attacks:	Interval between individual attacks	Empiric rating
Acute— ☐ Intermittent	☐ Long	Standard
Chronic—a. ☐ More or less continuous or recurrent wheeze	☐ Moderate	+25 to +50
b. ☐ More or less continuous with occasional acute exacerbations.	☐ Short	+50 to RNA

THERAPEUTIC RESPONSE

10. **Therapeutic Measures**				**Result**					
				Prompt	Satisfactory	Fair	Slow	Unsatisfactory	Poor
a.	Proprietary home remedies—name _____			☐	☐	☐	☐	☐	☐
b.	Desensitization—by what _____			☐	☐	☐	☐	☐	☐
c.	Anti-spasmodics	orally	atomizer	injection					
	☐ Adrenalin	☐	☐	☐	☐	☐	☐	☐	☐
	☐ Ephedrine	☐	☐	☐	☐	☐	☐	☐	☐
	☐ Aminophylline	☐	☐	☐	☐	☐	☐	☐	☐
d.	☐ Anti-histaminics	☐	☐	☐	☐	☐	☐	☐	☐
e.	Anti-biotics								
	☐ Penicillin	☐	☐	☐	☐	☐	☐	☐	☐
	☐ Streptomycin	☐	☐	☐	☐	☐	☐	☐	☐
f.	☐ Chemo-therapy	☐	☐	☐	☐	☐	☐	☐	☐
g.	☐ Morphine	☐	☐	☐	☐	☐	☐	☐	☐
	☐ Digitalis	☐	☐	☐	☐	☐	☐	☐	☐
	☐ Other Medicines— Specify_____	☐	☐	☐	☐	☐	☐	☐	☐
h.	☐ Psycho-therapy, change of climate, residence, or occupation, endocrines, etc. Specify_____				☐	☐	☐	☐	☐
i.	☐ Physiologic therapy—e. g. fever therapy, bronchoscopic ether and suction, rectal ether, intravenous alcohol, inhalation of oxygen, helium with oxygen while medication is reduced. Specify_____				☐	☐	☐	☐	☐

Table 2. Prognostic classification of asthmas based on degree of impairment of functional ability and character of response to therapy. Check applicable box.

Probable Etiologic Classification and Ratings	Degree of Severity	Degree of impaired functional ability	General Character of therapeutic response
Class I A(a) Standard	Mild	☐ Essentially no interference with normal activity	☐ Readily controlled by simple symptomatic therapy
Class I A(b) Substandard +25 to +75	Moderate	☐ Definite interference with activity but continued at reduced level	☐ Therapy definitely effective but larger doses and prolonged treatment required
Class I B,C +75 to RNA	Severe	☐ Pronounced interference with normal activity	☐ Controlled by considerable treatment
Class I B,C +75 to RNA	Very Severe	☐ Semi-invalid, or Status asthmaticus (invalid)	☐ Intractable Not adequately controlled by symptomatic treatment

Previously (2) we referred to the desirability of using response to therapy as a guide for separating asthmas into approximately four equal classes. The responses to treatment as reported by Coco and Cooke were 25 per cent excellent, 25 per cent good, 25 per cent fair, 25 per cent poor. It seems desirable to gather statistical data on this basis in order to evaluate its prognostic aid. In the meantime, lacking other reliable factual measures, we might use the M. I. R. studies as a very crude guide, and empirically charge a debit for the moderate to very severe types. The severity of attacks is proportionate to the quantitative relation existing between the sensitivity of the person and the amount of exposure to the asthmogenic agent. Etiology is the most important consideration in the proper classification for prognosis.

B *Mixed*—1—*SOME—STANDARD 2 TO 5 YEARS AFTER CURE. 2—DEPENDING ON FREQUENCY, SEVERITY AND RESPONSE TO THERAPY—SUBSTANDARD TO RISK NOT ACCEPTABLE. 3—SOME—STANDARD, DEPENDING ON AGE AND SEVERITY. 4—RISK NOT ACCEPTABLE. 5—GENERALLY—RISK NOT ACCEPTABLE, BUT MAY BE SUBSTANDARD 2 TO 5 YEARS AFTER CURE.*

Clinically common—unknown factor plus probable allergic etiology or primary reflex (non-allergic, non-infectious).

a. *Predominantly allergic—*

1—Extrinsic asthma, acute attacks, long periods of freedom—then suddenly more numerous and intervals shorter until attacks practically continuous.

2—Extrinsic in youth, free interval for years, then recurrence at age 50. No relation to occupational or environmental change.

3—"Asthmatic Bronchitis"—Dependent on colds. Characterized by wheezing, slight sensitivity to dust factors.

4—Most important group—Healthy all of life until onset at age 50. No allergy or previous asthma. No obvious cause; progressively more severe. Slight evidence of skin sensitivity, and positive family history in some.

5—Associated with severe chronic vasomotor rhinitis. Prognosis serious, majority women, onset about age 37. History chronic nasal trouble, first attack asthma of maximum severity. Course short—average four years. Apparently operation on sinuses predisposing cause in some, cure in others.

b. *Probably non-allergic, non-infectious (reflex)—*

Clinical types usually *obscure*. Etiologic groups: physical allergy, bronchial stenosis, psychosomatic reflex, acute left ventricular failure (cardiac asthma), emphysema with wheezing, and so-called intrinsic asthma, nasal bronchial reflex from foreign bodies or polyps, etc. Few cases lack element of atopy or infection. These types precipitated by heat, cold, effort, anxiety, nasal polyps, obstruction, or foreign body.

Wheezing only on exertion, preponderance of unilateral signs or symptoms, localized inspiratory stridor, and dyspnea, chiefly inspiratory. Relief by digitalization indicates cardiac asthma. Morphia ameliorates attack; administration in bronchial asthma invites death from asphyxiation. Physical and psychosomatic types amenable to therapy.

C *Infectious—GENERALLY—SUBSTANDARD TO RISK NOT ACCEPTABLE.*

Onset after active respiratory infection and fever, purulent nasal discharge, and cough with or without purulent sputum. Cough precedes and dominates the attack. Sputum purulent. Nasal mucosa red and swollen. Paucity of atopy in history and skin reaction striking. Troublesome exacerbations accompanied by fever and leukocytosis. Adrenalin not effective. Codein, with ephedrine or adrenalin more effective. Emphysema and bronchiectasis develop rather rapidly. Prompt cure of infection urgent. Aerosol antibiotics effectively abort. Allergic treatment not effective.

The following table of ratings for asthmas is based on the above etiologic outline. This schedule, of course, represents only my personal viewpoint.

SUGGESTED RATING SCHEDULE FOR ASTHMAS*

*SYMBOLS: NDR – No definite rating. RNA – Risk not acceptable.

CLASS I: KNOWN ETIOLOGY

		Action
A. **Primary.** (Bronchial Asthma) Mostly allergic etiology. Others are non-allergic reflex and may be classed as Idiopathic Asthma for rating purposes.		
Extrinsic		
Acute: May be	(1) **Seasonal** (generally most favorable)	standard
	(2) **Perennial** (less favorable: be alert for occupational and domestic dusts hazard)	NDR (mostly standard)

Chronic: More likely to be perennial, infrequently seasonal	NDR (mostly RNA)
Intrinsic Be sure of intrinsic classification, otherwise place in Class II. Likely to be perennial.	
Acute:	NDR (mostly standard)
Chronic:	NDR (mostly RNA)
B. **Secondary.** Classify and underwrite the primary underlying disease causing symptoms	NDR (mostly RNA)
C. **Mixed, Primary and Secondary**	sum of ratings for A and B +50

CLASS II: UNKNOWN ETIOLOGY

Idiopathic – Use analytic questionnaires for investigating etiology. If obscure etiology is reasonably cleared, reclassify as probable Class I A or B, and empirically add the following ratings according to the degree of certainty:

1. Very sure	standard
2. Fairly sure	+25 to +75
3. Believe right but uncertain	+75 to RNA

General Considerations:

Under age 15, standard may be given for best types only.
If age of onset is over 40, suspect Class I B, +30 for each decade over 40 (+25 to +100).

If overweight, increase build rating by one-half. If underweight, double rating for build.

Double indemnity may be granted to all acceptable for life coverage. Waiver of premium and disability income may be granted to standard risks according to the definition of what constitutes an acceptable claim. Applicants under age 40 are favorable if the qualifying waiting period is six months

and requires continuing total disability, and functioning ability and therapeutic response warrant a "mild" classification. Those in "moderate" classification may be considered for one and one-half to two times normal rate if time elapse since last attack is more than two years. One of the largest companies grants income disability, W. P., and D. I. with no debits for asthma up to their usual age limits of issue—50 for females, 55 for males—if risk is standard, attacks are "moderate", not more than twelve attacks a year, and last attack was four to six years past.

Asthma found on examination—rate on basis of history. The presence of wheezy breathing or scattered musical rales is not in itself a bar to standard consideration in Primary (bronchial asthma). The number of attacks per year in Primary (bronchial asthma) is not significant prognostically because the etiology is known and the attacks will continue to recur as frequently as the sensitized individual comes in contact with his specific asthmogenic incitant. The asthmatic attack is a temporary physiological disturbance and not organic. The attacks are self-limited, but treatment ameliorates and hastens restoration to normal. Do not penalize merely for taking treatment, medicinal, climatic or otherwise. Adequate and proper treatment of asthmas is best insurance against occurrence of chronic types and complications, such as the infectious respiratory diseases of bronchitis, pneumonia, emphysema, bronchiectasis, and the cardiovascular renal diseases.

DISCUSSION

Most of the present underwriting schedules of various companies are obsolete because their conception of the classification of asthmas is heterogeneous and not etiological. Consequently, their evaluation of symptoms and proper therapy are scientifically valueless for prognostic purposes.

Although the specific causes of asthma probably run into the thousands, this need not prove as bewildering as it might seem. For prognostic purposes, it is sufficient to simply classify any specific cause under its respective etiologic class.

Thus, asthma caused by sensitivity to ragweed, chicken feathers, pyrethrum, orris root, or lobster, all come under the common class of extrinsic allergy—a subdivision of Primary (bronchial) asthma. Asthma that is called perennial, and ascribed specifically to cat fur or house dust, is similarly classified. When ascribed to nasal polyps, it is intrinsic—another subdivision of Class I A. Primary asthma, complicated by infection, becomes Secondary and thus falls into the Mixed, Class I A and B, and the prognostic rating for the respective impairments is applied. When asthma is merely described as a chronic wheeze and the history is otherwise negative, we place it in Class II (Idiopathic), and we classify it as probably being a mixed clinical type—an unknown factor plus the probable predominant allergy, the suggested action for which is indicated. An asthma of unknown cause, associated with preceding respiratory infection and fever, also suggests the use of this same chart and the likelihood of its being a special clinical group C, infectious type, the suggested rating for which is indicated.

Although it is unlikely that comprehensive etiologic inquiries will be made, except for the larger or special risks, their use will depend largely on the reliability of information given on the routine examination. In most cases, the routine examinations should be sufficiently informative to permit action according to the probable etiologic classification without further investigation.

SUMMARY

1. Allergy is the most common cause of asthma and does not adversely affect life expectancy. It has been estimated that about 50 to 60 per cent of the population have minor manifestations, and that perhaps about 10 per cent suffer from a major form of allergy. The incidence of asthma among our population is estimated to be between 0.5 and 2 per cent.

2. It is probable that among asthmatics applying for life insurance, more than 95 per cent will be of the Primary (bronchial) class, and these as a class seem to have a normal life expectancy, except in chronic types.

3. Idiopathic asthmas that cannot be resolved into any known etiologic class, after investigation by questionnaires, should be rated empirically on the age of onset, severity, duration, and time elapsed since last attack.

4. All chronic asthmatics are usually uninsurable, but where there is a history of apparent recovery from the chronic state they should be critically appraised by the questionnaires.

5. All applicants over age 40, regardless of class, should also be appraised by questionnaire.

6. People rarely die of asthma. However, life insurance studies of asthmas disclose a significant percentage increase of deaths due to organic heart disease and other types of respiratory disease. It is reasonable to expect our mortality experience to improve by more efficient medical selection based upon the etiologic classifications. Asthma occurs for the first time before the age of ten in over two-thirds of all cases. Therefore, the appearance of asthma after age forty should be regarded with suspicion, as it may seriously affect life expectancy.

7. Because of the confusion in nomenclature and etiology, the selection of asthmatics will continue to be an underwriting problem. This will require the applied specialized knowledge of medical directors in the selection of asthmatic risks.

8. Modern therapy, with a better appreciation of the pathogenesis involved, including desensitization, immunization and avoidance, antibiotics, and chemotherapy, as well as symptomatic and antihistaminic therapy, tends to diminish the incidence of serious sequelae. The very recent control of the infectious group by antibiotic and chemotherapy may prove very significant in the prognosis of a class heretofore regarded as serious. It is reasonable to expect that the advent of this treatment will postpone both the incidence and effects of fatal complications, thus increasing longevity.

DR. SIMMONS—As we can all see, Dr. Schwarz has some very definite ideas on this subject of asthma. In fact, I am not sure that everyone here is going to agree with those very positive ideas. Are there questions or discussion of this important subject?

DR. ALBERT SEATON—How would the speaker differentiate as to prognosis of these various types; which ones are going to get worse and which ones are going to improve?

DR. SCHWARZ—I am very glad that question was asked, because it opens up a source of difficulty and one that has caused considerable confusion among our medical directors.

When you speak of which asthma is going to get worse, then you mean only one type of asthma—bronchial asthma. We must be clear on that, even at the risk of reiteration. Sometimes we understand what is said by different words in different ways. We think we understand what the speaker is saying and yet we do not. Which type of asthma is going to be confused? Certainly that type we are dealing with now, the acute type, which is generally accepted as a standard risk. That type is going to get worse which will not have a change in environment and will not be treated. Therefore, our tables as they are now constituted and applied are erroneous because we are penalizing the man who is being treated, or the man who goes away from the environment producing the asthma.

The extrinsic type, the very large group of primary asthmatics, is the result of environmental conditions—occupational dust, dust about the house, cat fur, the feathers in pillows, and other things of that sort. The minute the case is classified as primary asthma on an etiologic basis, disregard all other things. Thereafter, severity is of no concern because the severity of a response is directly proportionate, quantitatively, to the amount of asthmagenic agent present. Asthmatic attacks are bound to recur.

DR. SIMMONS—Are there any other questions? Thank you, Dr. Schwarz.

In closing this symposium, I wish to thank each member who has prepared material for discussion, as well as those who participated from the floor. I am sure that we all feel that such participation is an important part of any forum.

PRESIDENT ROBINSON—Dr. Simmons, we appreciate very much your excellent conduct of this important forum.

REFERENCES

1. Osgood, Robert B.: Aspects of Orthopedic Surgery, Preventive Medicine in Medical Practice, Chap. 25, 3rd Ed., N. Y. Acad. of Med. Comm. on Pub. Health Relations, 1942.
2. Karpinos, B. D.: The Physically Handicapped, Public Health Reports, Vol. 58, No. 43, Oct. 22, 1943, p. 1573.
3. Hitchcock, H. H.: Spondylolisthesis: Observations on Its Development, Progression and Genesis, J. Bone and Joint Surgery, 22:1–16, Jan., 1940.
4. Ghormley: Backache—Examination and Differential Diagnosis, J.A.M.A., 125:412–416, (June 10) 1944.
5. Mixter, Wm., J., and Barr, Jos. S.: Rupture of the Intervertebral Disc, with Involvement of the Spinal Canal, New England Jour. Med., 211:210–215, Aug. 2, 1934.
6. Hyndman, O. R.: Pathologic Intervertebral Disk and Its Consequences, Arch. of Surg., Vol. 53, No. 3, Sept. 1946, p. 247–297.
7. Cobb, John R.: Treatment of Scoliosis, Conn. State Med. Jour. 7–1943, p. 467–471.
8. vonLackum, W. H.: Surgical Treatment of Scoliosis, Surg. Treat. Motor Skeletal System, Part 1, Philadelphia, Lippincott, 1945, p. 146–198.
9. Key, J. A.: Penicillin and the Sulfonamides in the Treatment of Osteomyelitis and Pyogenic Arthritis, Bull. N. Y. Acad. Med., 21:87–98 (Feb.), 1945.
10. Wilflingseder: Penicillin in Hematogenous Osteomyelitis, Wiener Klinische Wochenschrift, 60:105, Vienna, 1948.
11. Fontaine and Grosse: Strasbourg Medicale, 109:1–10, 1948.
12. Orr, H. W.: Early Healing in Chronic Osteomyelitis and Compound Fractures, J.A.M.A., 128:1045 (Aug. 4), 1945.
13. Coleman, H. M., et al.: Cancellous Bone Grafts for Infected Bone Defects, a Single Stage Procedure, Surg., Gynec. & Obst., 83:392–398, (Sept.), 1946.
14. Prigge, E. K.: J. Bone & Joint Surg., 28:576, 1946.
15. Stark, W. J.: The Use of Pedicled Muscle Flaps in the Surgical Treatment of Chronic Osteomyelitis Resulting from Compound Fractures, J. Bone & Joint Surg., 28:343–350, (Apr.), 1946.
16. Graham, F. M., et al.: Penicillin in the Treatment of Chronic Osteomyelitis, Proceedings of the Staff Meetings of Mayo Clinic, Vol. 22, No. 26, Dec. 24, 1947, p. 589.
17. McClintock, J. A.: Reconstruction Surgery, J. Indiana Med. Assoc., 39:436, (Sept.), 1946.
18. Dublin, L. I., Jimenis, A. O., and Marks, H. H.: Factors in the Selection of Risks with History of Gallbladder Disease, Transac. Assoc. Life Ins. Med. Dir. of Amer., Vol. XXI, New York, Recording & Statistical Corp., 1934, p. 34.

19. Dublin, L. I., and Marks, H. H.: Mortality of Risks with Asthma, Transac. Assoc. of Life Ins. Med. Dir. of Amer., Vol. XX, New York, Recording & Statistical Corp., 1933, p. 191–214.

20. Schwarz, Berthold T. D.: Prognosis in Bronchial Asthma, Chronic Bronchitis and Bronchiectasis, Proceed. Med. Sec. Amer. Life Convention, Chicago, 1940, p. 98–156.

21. Rackemann, Francis M.: The Two Kinds of Asthma, Transac. Assoc. Life Ins. Med. Dir. of Amer., Vol. XXVII, New York, Recording & Statistical Corp., 1940, p. 69–83.

22. Rubin, Eli: Diseases of Chest, Philadelphia, Saunders, 1948, p. 442–450.

23. Taft, L.: Bronchial Asthma: A Critical Review, J. Allergy, 17:27, 1946.

24. Editorial: Classification of Asthma, J. Allergy, 16:199, 1945.

PRESIDENT ROBINSON—Last fall, I had the pleasure of visiting Dr. Rusk and visualizing the wonderful work he and his associates are doing in the Department of Rehabilitation and Physical Medicine at the New York University College of Medicine. He has kindly offered to have any of our membership who are interested make a personal tour of his department, whether that be later this afternoon or on some other occasion when you are in New York City.

During the last war, Dr. Rusk served as Chief of the Army Air Forces' Convalescent Service Division with the rank of colonel, and was awarded the Distinguished Service Medal in 1945. At present, he is consultant in medical rehabilitation to the Medical Director of the Veterans' Administration and to the secretariat of the United Nations. I present to you Dr. Howard A. Rusk who will talk to us on "Dynamic Therapeutics in Chronic Disease Pays Dividends."

DYNAMIC THERAPEUTICS IN CHRONIC DISEASE PAYS DIVIDENDS

HOWARD A. RUSK, M. D.
Professor and Chairman
Department of Rehabilitation and Physical Medicine, New York University College of Medicine, New York City

One of the most significant advances made in rehabilitation during the war and the immediate postwar period has been the increased recognition given to medical rehabilitation as an integral part of medical care. Just as satisfactory job placement is the capstone of any successful program of services to the handicapped, medical rehabilitation, starting at the earliest possible moment following acute illness or injury, is the foundation, since all subsequent rehabilitation processes are built upon the residual disability which medical care cannot eliminate.

Unfortunately, until the advent of World War II, medical care, psychological problems, and vocational rehabilitation were too frequently considered as separate and distinct processes having little relationship to each other. That they are interdependent and inseparable has been demonstrated by the successful programs in military and veterans' hospitals; also they have been recognized in civilian rehabilitation by the Barden-LaFollette Amendment, which expanded the federal-state vocational rehabilitation programs to include physical restoration, psychiatric services, and medical care as well as vocational guidance and training.

Extent of the Problem

Although the focus of attention has been centered on the disabled veteran, the extent of disability among our civilian population is far greater. There were 19,000 amputations during World War II, but over 120,000 major amputations

during this same period among our civilian population. Approximately 1,500 men were blinded while in military service during the last war, but 60,000 civilians lost their sight during this period. Some 265,000 men were permanently disabled as a result of combat injuries during the war, but 1,250,000 civilians were permanently disabled by disease and accidents in the corresponding four years.

There are some 23,000,000 persons in the United States handicapped to some extent by disease, accidents, maladjustment, or war. One-third of all draftees were rejected as unfit, and more than 1,000,000 had to be discharged shortly after induction. In 1946, 10,400,000 persons suffered disabling accidents, and of these, 370,000 were disabled permanently. It is estimated that there are over 7,000,000 persons in the United States disabled by diseases of the heart and arteries, 6,850,000 from rheumatism and arthritis, and 2,600,000 from orthopedic conditions.

These are the numbers, but they cannot tell the story of pain, anxiety, suffering, and all of the vital secondary problems that disease and disability leave in their wake. Aside from pain and tearing personal and family anguish, the economic costs of disease and disability are staggering.

Toll of Chronic Disease

Today, as medical science moves forward in the prevention and cure of infectious disease, chronic illness has become the nation's primary medical problem. In 1900, seven chronic diseases (cancer, diabetes, intracranial lesions of vascular origin, diseases of the heart, diseases of the arteries, cirrhosis of the liver, and acute and chronic nephritis) were responsible for 25.7 per cent of all deaths in New York State. Between 1900 and 1940, a period in which the population of New York State increased by 85 per cent and the total number of deaths increased by only 13 per cent, the number of deaths from this group of chronic diseases increased by 200 per cent and now includes over two-thirds of all deaths in the state.

Whereas diabetes ranked twenty-seventh and arteriosclerosis thirty-fourth as causes of death in 1900, they were eighth

and tenth in 1944. Added to this is the tremendous long-term toll of chronic illness and disability. Acute infectious disease usually results in rapid recovery or death, but chronic illness may linger for years. Its social and economic costs cannot be measured by mortality tables alone.

One of the principal causes of the increasing prevalence of chronic disease has been the great advances in medical and surgical care which have prevented death and produced an aging population. Two thousand years ago, the average length of life was twenty-five years; at the turn of the century it was forty-nine; today, it is sixty-six. In 1900, one person in 25 was sixty-five years of age or older; it is estimated that in 1980, the ratio will be one in ten. The chances are now two out of three that a young man now starting his working life at the age of eighteen, will live to his retirement age of sixty-five. A forty-five year old man today has 70 in 100 chances of reaching the age of sixty-five, and the chances for a fifty-five year old man are 78 in 100. White men now at the age of sixty-five can expect to live an additional twelve and one-half years on the average, and white women an average of fourteen and one-quarter years.

As people become older, their medical needs change, and they demand more medical service. In 1940, the 26.5 per cent of the nation's population over forty-five required over half the nation's medical services. By 1980, it is expected that the number of persons over 45 will constitute nearly half of the population.

The incidence of chronic disease is definitely related to economic status. It develops most frequently among families of low income, and when it strikes, results in further reduced income and depletion of financial reserves. In the National Health Survey of 1936, chronic illness was found to exist nearly twice as much among persons on relief as those earning $3,000 a year and over. A more recent study in Illinois showed that 23 per cent of all public assistance recipients were chronic invalids. In Connecticut, the percentage was 30, and in New Jersey, 38 per cent of those receiving old age assistance are chronically ill.

As chronic disease is usually nonreportable, reliable statistics as to its extent are difficult to find. Most currently used figures spring from the National Health Survey conducted in 1935 in which 3,000,000 in 83 cities and 23 rural areas in 19 states were surveyed. It was from that report that the figure 23,000,000 was derived. Later reports, based on smaller samplings, however, have indicated that chronic disease is even more prevalent. In a recent survey in New Haven conducted by the School of Public Health of the Yale University College of Medicine, it was found that 121 persons for each 1,000 in the population suffered from chronic illness, and that one-third of this number were totally disabled.

Although chronic disease is more prevalent in older persons, authorities state that 16 per cent of all persons with known chronic disease are under twenty-five years of age. The Yale study found the percentage to be 33.

Rehabilitation Pays Economic Dividends

The present programs of the military services and the Veterans' Administration have dramatically demonstrated that rehabilitation pays economic as well as social and personal dividends, even for those who suffer from long-standing chronic illness. Typical of the results obtained in the Veterans' Administration Medical Rehabilitation Service is a study of 130 chronic neurologic patients in one hospital, all but two of whom were World War I veterans, and many of whom had not been out of bed in ten years. After nine months of medical rehabilitation, 25 had left the hospital and were employed; 40 others had been discharged to their homes capable of light work, and, of those remaining, 30 were ambulatory and undergoing advanced rehabilitation and 25 were capable of some self care. All but ten of the group had shown worthwhile permanent improvement. With a five-year life expectancy of these patients, and a per patient day hospitalization cost of over $12.00, rehabilitation of this one group has saved the government, and eventually the taxpayer, over $1,250,000. It would seem logical that a similar program for the civilian chronically ill would result in comparative savings.

The experience of the Federal Office of Vocational Rehabilitation has also shown how rehabilitation pays off economically. During 1944, 43,997 persons underwent rehabilitation under the joint federal-state vocational rehabilitation programs. Of this group, 22 per cent had never held jobs, and 90 per cent were not employed at the time they started their rehabilitation. Their average annual wage after rehabilitation was $1,768, as compared with $148 before. Many had been on public assistance, at a cost of $300 to $500 annually, but the cost of their rehabilitation was but $293 per case, a single rather than annually recurring expenditure.

Rehabilitation in the General Hospital

Although it would seem logical that medical rehabilitation would be an important service in every civilian hospital, there has been little or no attempt, until recently, to establish such programs in civilian hospitals. Of the 1,425,222 hospital beds in the United States in 1947, 42 per cent were in general hospitals, but these 592,453 beds cared for over 92 per cent of all patients. Rehabilitation, in varying degrees, has been available in some tuberculosis, mental and other specialized hospitals, but little provision has been made for dynamic convalescent care and rehabilitation of the over 14,500,000 persons who are patients in general hospitals each year.

The first comprehensive total medical rehabilitation program in any community hospital in this country has recently been inaugurated at Bellevue Hospital in New York. Operated under the professional direction of the Department of Rehabilitation and Physical Medicine of the New York University College of Medicine, the service has bed facilities for 80 patients, and offers a program of physical medicine, physical therapy, occupational therapy, corrective physical rehabilitation, social service, corrective speech, psychologic services, vocational guidance, education, and planned recreation. It operates as a service department to the other departments of the hospital in much the same manner as the x-ray and laboratory, and treats both in-patients and out-patients on reference from the other services of the hospital.

The rehabilitation service in Bellevue Hospital, which will be enlarged to 600 beds when the construction planned is completed, is the first step in a plan by the Department of Hospitals of the City of New York to provide all patients in municipal hospitals of the city with medical rehabilitation services. The extent to which rehabilitation has entered into future planning in New York in both public and private hospitals is shown by a recent report of the Hospital Council of Greater New York, in which it was suggested that 25 per cent of the bed capacity of the city's general hospitals should be allocated for convalescence and rehabilitation. This would mean one such bed for each 1,000 of the city's population.

The interest in extending medical rehabilitation services in general hospitals is not limited to New York City or other large urban areas. The Veterans' Administration has recently established such services as major departments with specified bed allocations in all Veterans' Administration Hospitals. The New York State Health Preparedness Commission has recommended that the State should build or acquire and support a chronic disease hospital center contiguous to a general hospital, in close proximity to a medical school, and staffed and operated by contract with such hospital and medical school in each of five geographic regions of the State. They have also recommended that "rehabilitation services for the disabled and chronically ill be developed as rapidly as possible in selected general hospitals throughout the State by providing the services of trained personnel and by providing funds for the training of needed personnel in this field."

Today, throughout the nation, there are some 150 communities which have expressed interest in establishing rehabilitation services either independently or in conjunction with existing hospitals, medical schools, or community agencies.

The practice of rehabilitation for the general practitioner, or for any physician, begins with the belief in the basic philosophy that the doctor's responsibility does not end when the acute illness is ended or surgery is completed; it ends only when the individual is retrained to live and work with what is left. This basic concept of the doctor's responsibility can

be achieved only if rehabilitation is considered an integral part of medical service. Any program of rehabilitation is only as sound as the basic medical service of which it is a part. The diagnosis and prognosis must be accurate, for it is upon them that the feasibility of retraining is determined.

In addition to the general diagnostic studies, the medical evaluation of the orthopedically handicapped must include muscle tests, joint range of motion, and tests for the inherent needs in daily living. In the Rehabilitation Service at Bellevue Hospital, a check list of 96 items is used to determine these factors. They include: first, bed activities, such as moving from place to place in bed, and the ability to sit erect; second, toilet activities; third, eating and drinking; fourth, the ability to dress and undress, such as tying shoe laces, manipulating buttons, zippers and other fasteners, and applying and removing braces; fifth, hand activities, for example, winding a watch, striking a match, and using various door knobs and latches; sixth, wheelchair activities, getting from the bed to the wheelchair, the wheelchair to the bed, and in and out of the bathtub; and finally, elevation activities, which include the needed abilities for walking, climbing and traveling.

At first glance, such a test list sounds formidable and time consuming, but in reality, the information may be easily obtained by a therapist, nurse, a well-trained volunteer or a member of the patient's family. From the special check sheets used for charting the activity accomplishments, information is readily available both on the status of the patient at the time of admittance, and his progress while undergoing rehabilitation.

The use of such a check list is particularly helpful if personnel are not available to do definitive muscle testing and accurate range of motion determination, for the daily activities test can be completed in the hospital, the physician's office, or in the patient's home. The subsequent training program is designed to teach the patient the various skills and activities which he cannot perform.

In Bellevue Hospital, after the basic medical workup, the range of motion, muscle and needs of daily living tests, the

physician in conference with the therapists and other staff members, prescribes for the patient a full day's program running from nine in the morning to four in the afternoon. These prescribed activities include training in the ambulation and elevation rooms, remedial gymnasium, occupational therapy, physical therapy, speech therapy, or any other activity which may be helpful in meeting the specific needs of the patient.

This same approach is being followed in the new Institute of Rehabilitation and Physical Medicine which recently opened as one of the first operating units of the new New York University-Bellevue Medical Center. Patterned after the recommendations of the Baruch Committee, the Institute offers a complete program of physical medicine and rehabilitation, including physical rehabilitation and retraining, psychosocial adjustment, and vocational evaluation and guidance for physically handicapped persons. It provides an integrated program of physical therapy, occupational therapy, physical rehabilitation, social service, vocational guidance and testing, and recreation, designed to teach the patient who has a physical disability "to live within the limits of his disability, but to the hilt of his capabilities". With facilities to care for an in-patient census of 30 and daily out-patient load of 100, the Institute places special emphasis on retraining in elevation, ambulation, crutch-walking, and the physical skills necessary for the activities inherent in daily living.

The Institute's service and facilities are available to any patient, either on an in-patient or out-patient basis, who in the opinion of the referring physicians and the Institute's staff, could benefit from rehabilitation and retraining. It is expected that the majority of cases will be those needing definitive physical medicine or rehabilitation as the result of injuries, arthritis, orthopedic and neurological disabilities, and diseases of the heart and arteries. Special facilities are available for a limited number of paraplegics and hemiplegics. Although the Institute is not prepared to do vocational training of the handicapped *per se*, special workshops are available for vocational evaluation and guidance and job hardening under medical supervision for arrested tuberculous and cardiac

patients. Complete vocational and guidance services are available for all types of persons with physical disabilities.

One of the special features of the Institute is the Prosthetic Service in which consultation and recommendations are made on the selection, fitting and adjustment of prosthetic devices of all types, and intensive training is given in their use. The Institute does not manufacture or sell prosthetic devices, but has skilled technicians available for services for all types of amputations, and for the minor adjustments and refittings frequently necessary to make artificial limbs more comfortable and efficient to the wearer.

Although we have in this country the finest institutions in the world for definitive medical care and for vocational training, outside of the military services and the Veterans' Administration there are but a small handful of civilian agencies and organizations equipped to provide for the patient with a physical disability the necessary retraining in physical skills which are a requisite for later vocational training. The physician in the past has thought too much about the physiological and clinical aspects of the patient's disability. The vocational counselor too frequently has thought only in terms of physical skills which could be utilized vocationally. Between the two, however, there is a wide area through which most physically handicapped persons must go when their definitive medical care is completed but before they are ready to undergo vocational training. In this area lies the physical retraining in skills necessary for the carrying on of the activities inherent in daily living and common to all types of work.

Except in a few isolated instances, the physically handicapped person must be retrained to walk and travel, to care for his daily needs, to use normal methods of transportation, to use ordinary toilet facilities, to apply and remove his own prosthetic devices, to communicate either orally or in writing. These are such simple things that they are frequenty overlooked, but the personal, vocational and social success of the handicapped person is dependent upon them.

Age plus physical disability will prevent many chronically disabled patients from returning to competitive employment. Vocational placement, however, is not the only valid goal of rehabilitation. The factors of self-care and the ability to do productive work while still living in a hospital, home or other adult institution, are also valid objectives. They are valid medically and socially, for their effect on the well-being of the individual, and economically, in that the personnel and operating costs of the institution or the patient's home are thereby reduced. Using the known techniques of selective placement, these individuals should be allowed to work within the limits of their capacities. Work, properly prescribed, is one of the most valuable therapeutic tools in the treatment of the geriatric patient and those suffering from chronic disease. The problems of chronic disease and the aging population can be met only by the creation and utilization of abilities, rather than the building of facilities.

Because of the close inter-relationship between physical disability and insurance, there are special implications in the rehabilitation program for those physicians who are concerned with insurance, particularly in the field of accident and disability insurance. Each year some 90,000 workers in the United States are permanently disabled as the result of industrial accidents. Over 2,000,000 other workers suffer temporary disabilities from accidents. Total work time lost by these men in 1947 was 280,000,000 man-days, or the equivalent of 1,000,000 men kept out of work continuously for more than a full year.

Many of these men, however, have no chance for rehabilitation and the opportunity to return to productive work. In most cases, those with serious physical disabilities are retired on disability compensation, physical and psychological cripples, their earning power reduced to zero, and their buying power reduced by 50 per cent, unwilling liabilities to themselves, to their families and to the nation. Added to this tremendous social waste are the direct costs of over $2,600,000,000 a year in wage loss, expenses for medical care, overhead costs of workmen's compensation, damaged equip-

ment and materials, production slowdown, and time lost by fellow-workers. Industry has a tremendous stake in reducing physical disability. Millions of dollars are spent annually on prevention, the first phase of medicine, through accident prevention and health maintenance services. The amount spent on the second phase of medicine, treatment, is shown by the fact that doctors and hospitals in New York State alone last year received more than $25,000,000 for services rendered injured workers in the nearly 800,000 reported cases of industrial accidents and disease. But thus far, rehabilitation, the third phase of medicine that takes the patient from the bed to the job, has been neglected.

In rehabilitation, as in definitive medical care, the general practitioner is an essential and integral member of the therapeutic team. Today, as new and mounting demands are made upon him to restore his patients to maximum economic and social effectiveness, he must turn to the expanding field of rehabilitation and physical medicine for increased technical skills and assistance. However, many of these simple techniques he can apply directly in his office, home and hospital practice to help his physically disabled patients "learn to live and work with what they have left."

PRESIDENT ROBINSON—Thank you, Dr. Rusk. We are deeply indebted to you and grateful that you brought with you so many of these marvelous patients, with such wonderful rehabilitative results.

Dr. McLeod C. Wilson, Medical Director of The Travelers Insurance Company, will now present a paper on "Ulcers of the Duodenum."

ULCERS OF THE DUODENUM

McLeod C. Wilson
Medical Director
The Travelers Insurance Company, Hartford, Connecticut

Ulcers of the duodenum have been discussed before this Association at various times over a considerable number of years. The approach has been largely clinical, with the etiologic theories and the medical and surgical treatment being emphasized. There is no need, therefore, for me to go into this aspect as it is familiar to all of you. However, an analysis of ulcer cases from an insurance statistical viewpoint has not been made recently. I shall attempt to show that insurance statistics are garbled because of the lack of uniformity in accurately reporting the location of the ulcer. Also, the rates have been predicated on the number of attacks and the time elapsed after the last attack. The meaning of "attacks" is based on criteria of a very vague nature. We must accept the fact that ulcers are not cured but rather remain quiescent for varying periods of time. Dr. Bockus (1), in the Journal of the American Medical Association, January 31, 1948, makes these comments as to what is meant by "cure": "Nearly 75 per cent of patients who are adequately treated medically for duodenal ulcer, including advice for the prevention of recurrences, remain sufficiently well so that they suffer no economic loss and are soon relieved of their recurrences of minor distress by resuming at once a strict regimen for a brief period of time." This author also states that only about 10 per cent of patients with duodenal ulcer get into any really serious difficulty. He states there is no present therapeutic agent which guarantees against recurrence of ulcer even though it be continued throughout life. It is also interesting to note that even without any symptoms at any time, duodenal ulcers may be demonstrated. Dr. Engel of Philadelphia, in an article in the Journal of the American Medical Association of November

15, 1947, reports the findings of St. John (2) and his colleagues on fluoroscopic and x-ray examinations of the stomach and duodenum on 2,413 individuals who were considered normal and had never had any gastric complaints at any time. This study revealed the presence of 3 malignant growths, 5 gastric ulcers, and 54 duodenal ulcers.

Fear of the possible dramatic episode has colored our thinking and has contributed to the lack of uniformity in underwriting practices. Before this Association in October, 1946, Dr. Crohn (3) presented a paper which gave very favorable statistics and should help to offset some of our apprehension. Hemorrhage and perforation are the two major complications of duodenal ulcer, and even in this impaired group the mortality is less than 1 per cent. The discussion from the floor gave some indication of skepticism. In our desire to avoid adverse mortality experience, we read the available material written by clinicians, surgeons, and roentgenologists. Recently, a number of articles on the subject have appeared in the Journal of The American Medical Association, some giving mortality rates that without further analysis appear alarming. However, this was a group of seriously ill patients, hospitalized, and constituted 10 per cent, or less, of all ulcer cases. From an insurance standpoint, the mortality of the present group of cases is not unfavorable, and I believe this confirms Dr. Crohn's clinical experience. I trust that these facts will offset any unwarranted fear of the possible grave developments, for the group as a whole does not reveal these as probabilities.

The incidence of ulcer of the stomach and duodenum can only be estimated. Dr. Ivy has estimated that 1,500,000 persons in the United States over ten years of age are affected by peptic ulcer during a given period of ten years. This is about 10 per cent of the population. Zetzel reported that in an Army Station Hospital with an admittance record of 21,856 patients, there was an ulcer incidence of 7.2 per cent. Hurst concluded as early as 1929, from a study of autopsy statistics, that it may be assumed about 10 per cent of all individuals have ulcer of the stomach or duodenum at some time during

life. Dr. Ralph C. Brown (4) of Chicago commented that this might be accepted as a conservative estimate. A large insurance company reported an incidence of 7 per cent plus, among their employees.

In attempting to visualize, numerically, the incidence of the disease, we must keep in mind that those under medical observation or treatment constitute a very small percentage of the whole. Diagnosis is often delayed because attention has become focused on associated symptoms, such as spastic colon, gallbladder dysfunction, irregular peristalsis, etc. The number that goes undiagnosed must be tremendous, because vague histories or symptoms are often disregarded by the individual or his physician, and his complaints are attributed to this, that, or the other thing in his troubled life. Self-treatment can be gauged to a degree by the fact that manufacturers of antacid mixtures find it profitable to sponsor radio programs and advertise extensively in the lay journals. A broad visualization of the incidence, which would include the active, quiescent, and potential cases, must be somewhere between 7 and 12 per cent of the adult population. Statistically, we are, therefore, limited to a study of the few who because of environment, severity of their symptoms, or complications have adequate medical investigations.

It is factual that the majority of ulcers first appear in the age group 30–40(5). Most companies issue 50 per cent of their business at age 35 or under (The Travelers is 45 per cent). Therefore, it is only reasonable to assume that, predicated on the incidence of the disease, 10 per cent of the exposed group will develop ulcers subsequent to issue. Those cases placed in substandard classifications are probably reduced by two-thirds within the select period by removal of ratings according to the underwriting practices of individual companies; thus a further volume of ulcers, actual or potential, is added to the exposure. It, therefore, is only reasonable to conclude that whatever the actual mortality may be for ulcers of the duodenum, it must be minor.

It is also factual that loss ratios in the past fifteen years have shown 33 per cent improvement that would have been

reflected otherwise, because of the disease incidence, if the impairment were important. Confirmation of an improving mortality experience may be found in the Transactions of The Actuarial Society of America (6), Vol. XLVIII, Part 2, No. 118, October 1947. This is an analysis of the Joint Mortality Experience of sixteen of the larger life insurance companies. This study was compiled by Mr. James S. Elston (6), Assistant Actuary of The Travelers Insurance Company, and the figure obtained from Table 11. Deaths directly due to duodenal ulcer in The Travelers have been for many years rather consistently under .3 per cent. The vital statistics for the State of Connecticut (7), compiled for the year 1943, showed 57 deaths from ulcers occurring in a total of 19,408 deaths, or .29 per cent (combined duodenal and gastric .71 per cent). The figures for New York City (8) in 1945 gave a combined total, gastric and duodenal, of .9 per cent, the duodenal portion being .3 per cent.

Predicated on the above, it would appear that the duodenal ulcer group could be absorbed without materially affecting company mortality rates. In addition, the figures which will be shown later seem to warrant placing the duodenal ulcer in the same category as the automobile and passenger aviation hazard, or the common cold with its complication potentials.

Dr. Lahey (9), in his discussion of Dr. Dublin's (10) paper in 1935, is quoted as follows: "The percentage of gastric ulcer as compared with duodenal ulcer, as presented in Dr. Dublin's statistics, I think must be wrong, because the examining doctors have not made accurate diagnoses. We have had 190 gastric ulcers out of this 2,100, and that has been about the average. We average from one to nine to one to ten gastric to duodenal." Zetzel found in the military age group a ratio of 25 duodenal to 1 gastric. St. John, in his study of the asymptomatic group, reported 11 to 1. With such figures constantly being reported, it is no wonder that clinicians and surgeons question insurance statistical material.

In an attempt to have an experience analyzed by the Actuarial Department, it was quickly discovered that the incidence of gastric ulcers was out of all proportion to the clinical

figures. It was then necessary to review the actual files in a sufficient number of cases to determine what was wrong. This study furnished the key to the problem and proved beyond any question of doubt that the trouble lay at the door of the physician and surgeon because of the loosely used terms of "peptic," "gastric," and "stomach" ulcers. Then, to a large degree it appeared that the insurance companies had accepted as fact the applicant's statement to the examiner as to the nature of his trouble. It was noted that when a diagnosis of duodenal ulcer was given, it was usually correct, but when the diagnosis of stomach or gastric ulcer was given, it was rarely correct—an estimated error of 20 to 1. This indicated why the Medico-Actuarial figures on stomach ulcers were not too unfavorable, the fact being that this experience was modified by the inclusion of more than 50 per cent of the more or less benign duodenal ulcers. Further proof of this comes from the report of Allen and Welch (11), in the New England Journal of Medicine, dated April 22, 1948, in which they review gastric ulcers observed at the Massachusetts General Hospital from 1931 to 1940. This showed that 14 per cent of ulcers first considered benign were later proved to be malignant.

From the above comments, it was obvious that a mortality study predicated on the recorded information in the files of life insurance companies would be worthless. Therefore, it seemed plausible to study disability claims, as here at least was a group in which the case history could be followed to its termination. The disability requirement was three months' total disability before insurance benefits became operative. We are, therefore, dealing with a group of the more serious cases, in contradistinction to the vast number which we might term "run of the mill". The group constitutes part of the 10 per cent of patients with duodenal ulcer who get into serious difficulty. It was very apparent that the disease rarely produced prolonged total disability, as only 324 claims occurred among a total of 30,000 claims for all causes.

This study made it necessary to read every file in its entirety and tabulate the points that follow. One of the most difficult parts of the analysis was to determine which of the

claims were predicated on ulcers of the stomach and which on the duodenum. If we accepted the diagnosis as factual when the proofs of claim were received, 45 per cent of them would have been ulcers of the stomach. However, during the course of the claim, additional medical evidence received showed the ulcer to be of the duodenum in most instances.

The following analysis is of interest and may be useful in considering the background for future studies:

A. Claims terminated with insurance in force as of June 1948

		75% (242)
a.	Average age:	
	1. At time of issue	34 yrs.
	2. At onset of disability	42 yrs.
	3. Present	55 yrs.
b.	Ulcer location:	
	1. Stomach	6%
	2. Duodenum	94%
c.	History of ulcer (or suggestive) at time of application:	
	1. Admitted	10%
	2. Not admitted but developed in claim investigation	17%
	3. No signs or symptoms prior to issue	73%
d.	Average duration of symptoms prior to onset of disability:	12⅔ yrs.
e.	Period of disability:	
	1. Under one year	65%
	2. More than one year	35%
f.	Onset of disability:	
	1. Initiated by acute perforation	18%
	2. No prior history (silent)	3%

g. Age at time of perforation:

1. 20–30	5	12%	(43 cases)
2. 30–40	13	30%	
3. 40–50	17	40%	
4. 50–60	7	16%	
5. 60+	1	2%	

h. Onset of disability:

1. Initiated by hemorrhage	49%
2. No prior history (silent)	10%

i. Age at time of hemorrhage:

1. 20–30	9	8%	(118 cases)
2. 30–40	40	34%	
3. 40–50	48	41%	
4. 50–60	20	16%	
5. 60+	1	1%	

j. Operative cases:

1. Gastroenterostomies	48	44%	(135 cases)
2. Subtotal gastrectomies	31	29%	
3. Simple closures for perforation	29	27%	
4. Operations for obstruction or intractable symptoms	27	25%	out of total

B. Existing Claims—37 cases (37)

a. Average age:

1. At time of issue	36 yrs.
2. At onset of disability	43 yrs.
3. Present	58 yrs.

b. Average duration of disability 11 yrs.

c. Hemorrhage initiating disability 38%

d. Perforation initiating disability 8%

e. Operative cases 35%

f. Continuing cause of disability in 22 cases was for a disease other than ulcer 59%

g. Ulcer symptoms constituted 27%

h. Because of retirement and age 14%

C. Deaths—45 cases 14% (45)

a. Average age:

1. At time of issue 38 yrs.
2. At onset of disability 51 yrs.
3. At death 59 yrs.

b. Average duration of symptoms prior to disability 15 yrs.

c. Average time elapsed after issue 12 yrs.

d. Period of disability:

1. Under 1 year	22	49%
2. More than 1 year	23	51%

e. Stomach ulcers 6 13%

f. Duodenal ulcers 39 87%

(50% of the group were submitted as ulcers of the stomach which subsequent investigation proved to be duodenal in type.)

g. Hemorrhage the initial cause of disability—27 cases (27)

Age grouping:

1. 30–40	1	4%
2. 40–50	10	37%
3. 50–60	13	48%
4. 60+	3	11%

h. Perforation the initial cause of disability—9 cases (9)

Age grouping:

1.	30–40	1	11%
2.	40–50	3	33%
3.	50–60	3	33%
4.	60+	2	22%

i. Deaths by Cause:

1.	Cardiac	12	27%
2.	Cerebral apoplexy	5	11%
3.	Post operative	7	15%
4.	Ulcer of stomach	4	9%
5.	Ulcer of duodenum	1	2%
6.	Cancer of stomach	3	7%
7.	Cancer—other organs	4	9%
8.	Suicides	2	4%
9.	Other causes	7	16%

After the results were tabulated as shown, the Actuarial Department looked over the figures and worked out an estimated mortality on this group of disability cases.

On the 324 cases there was a total exposure of 3,664 policy years. The cases were entered at one age and included in the experience until there was an exit, or until 1948. The age at entry was taken as the difference in calendar years between the effective date of disability and the date of birth, and the age of exit as the difference between the year of exit, or 1948, and the year of birth.

A total of 47 deaths was found against an expected of 39, based on the most recent mortality table—the Joint Committee on Mortality 1939–1944. This produced a ratio of 121 per cent for the total exposure. Two more deaths are shown here than are recorded on the tables; these two extra deaths occurred since the files were reviewed but they are included in our actuarial statistics.

The exposure, of course, is not large enough to warrant too great refinement because the margin of error is so large, and so few policies are involved. Even so, in this group of more seriously impaired ulcer cases, disabled because of their ulcer, the finding of only 121 per cent mortality was indeed gratifying.

CONCLUSIONS

1. Duodenal ulcers comprise 89 per cent of the total. This appears to substantiate the large error in information obtained from applicants.
2. Histories are poorly taken, considered unimportant by the examiner, or withheld by the applicant.
3. The disease is essentially chronic in character and serious episodes usually occur only after many years of ulcer signs or symptoms.
4. Hemorrhage and/or perforation occurs most frequently in the 40–50 age group.
5. Histories of hemorrhage or perforation do not materially affect the mortality.
6. No malignancy occurred at the site of a duodenal ulcer—three cancers of the stomach followed ulceration—the balance were unrelated.
7. The average age of death was in the sixtieth year and in the twenty-first policy year.
8. The known ulcer cases on the books are infinitesimal compared to the unknown.
9. Disability claims are a valuable source of information in mortality studies.
10. The mortality in this study is 121 per cent.
11. To obtain an accurate Medico-Actuarial study, it will be necessary to start anew. If we are to differentiate duodenal from gastric ulcers, we must collect accurate information about the location of the lesion when this is available from sources known to be reliable.

REFERENCES

1. Bockus, H. L.: Gastro-enterology, J. A. M. A., Vol. 136, p. 293, 1948.
2. St. John, F. B., et al.: Experiment in Early Diagnosis of Gastric Cancer, Annals of Surgery, p. 119, 225, 1941.
3. Crohn, B. B.: Digestive Ulcers, Their Significance and Prognosis, Transactions of The Association of Life Insurance Medical Directors of America, Vol. XXX, p. 177, Recording & Statistical Corp., New York City, 1946.
4. Brown, R. C.: Ulcer of Stomach, Duodenum and Jejunum, Oxford System of Medicine, Vol. III, Part 1.
5. Rafsky, Henry A., et al.: Symposium on Peptic Ulcer, J. A. M. A., Vol. 136, p. 739–752, 1948.
6. Elston, J. S.: Actuarial Society of America, Vol. XLVIII, Part 2, October, 1947.
7. Connecticut Vital Statistics, 96th Registration Report, 1943.
8. Vital Statistics, City of New York, Department of Health, City of New York, 1941–1945 inclusive.
9. Alvarez, W. C. and Lahey, F. H.: Discussion of the Selection of Risks with a History of Gastric or Duodenal Ulcer, Transactions of The Association of Life Insurance Medical Directors of America, Vol. XXII, p. 50, Recording & Statistical Corp., New York City, 1935.
10. Dublin, L. I., et al.: The Selection of Risks with a History of Gastric or Duodenal Ulcer, Transactions of the Association of Life Insurance Medical Directors of America, Vol. XXII, p. 24, Recording & Statistical Corp., New York City, 1935.
11. Welch, C. E. and Allen, A. W.: Carcinoma of the Stomach, New England Journal of Medicine, Vol. 238, No. 17, p. 583, 1948.

* * *

PRESIDENT ROBINSON—Thank you for that very thoughtful presentation, Dr. Wilson. This paper will be discussed by Dr. H. Clive McAlister, Medical Director of The Lincoln National Life Insurance Company.

DR. H. CLIVE MCALISTER—I was asked to discuss some recent studies made by my company of its experience with ulcer disease. These studies are "predicated on the recorded information in the files of life insurance companies." If I understand Dr. Wilson's thesis, it is his opinion that such data are "worthless". I think he is wrong. In the first place I cannot join in his sweeping denial of any and all validity to any and all conclusions based upon study of the ulcer material we now have. Secondly, because I see no great hope of markedly improving the homogeneity of ulcer groups in the near future, I think the testimony of the past groups, imperfect

as they may be, is the best index of the mortality of future groups which unfortunately will suffer at least for a while from the same imperfections in about the same proportions and degrees. Dr. Wilson has conducted interesting observations among those policyholders of his company whose claim for disability for ulcer has been allowed. Undoubtedly the group contains the worst of living cases. On the other hand, these people claiming disability on account of ulcers are, in effect, annuitants, the mortality among whom is notoriously low. Also, I suspect that there is much neurosis among them, the effect of which is to reduce group mortality. Certainly the characteristics of the disability group are different from one made up of insurance applicants who have had ulcer.

Our ulcer investigation was part of a series of studies on our issues of 1931 to 1945 carried to policy anniversaries in 1946. The table we chose as representing standard mortality was that of Miller because it reflects the actual experience with a vast bulk of business issued standard in 1930–39, a period covering most of the years of our studies. As you know, Miller's Table is based upon the same cases and exposure as is the Commissioner's Standard Ordinary Table; but unlike the Commissioner's table, it includes a select as well as an ultimate period and it does not contain margins for the various contingencies. Although we have used a modern table, the actual experience of our company has been substantially more favorable. On business issued standard in 1934 and thereafter carried to 1944 anniversaries, the ratio to Miller's Table was about 68 per cent at age 20, increasing with remarkable uniformity to about 92 per cent at age 60. An overall ratio of about 80 per cent is a conservative and useful basis of comparison.

Although most of our series of studies concerned business originating in 1931 and subsequently, in some impairments we were curious as to group behaviour in the more remote years of the ultimate period. In the case of ulcer disease, we studied all issues during and since 1921.

We have made no distinction between business written by our own agents and that acquired through reinsurance and, ex-

cept as noted, we have included substandard (calculated upon a standard basis) with our issues. All cases complicated by other impairments are excluded, the exceptions being "dyspepsia and indigestion", "gastritis", "nervous indigestion", "diseases of the stomach not included elsewhere", and "duodenitis". You will recognize these as usually merely descriptive terms that are characteristic of and cluster about ulcer cases.

I wish to state one point about our studies and all others emanating from a single company—all cases included have been classified by the same underwriting department and in many, many instances by the same underwriters. During the twenty-five years involved, there have been occasional changes in the relevant rules of selection and in the schedules followed, but the views of the disease and its complications which guided the handling of individual cases, and the interpretations placed upon the commoner forms and the varied language in which information concerning ulcer comes to us, have been uniform. This means a substantially higher degree of group homogeneity than is possible with material combined from multiple sources. I have included mortalities only when at least ten deaths have occurred. On all the displays dealing with policy duration, the first three years are grouped together because according to modern concepts this is the select period. In each instance, however, I have also grouped the first five years possibly as a gesture toward tradition but also in the hope that such arrangement may aid in comparing these with prior results.

DUODENAL ULCER—NOT OPERATED

0 – 2 years from last attack

Policy Years	Deaths	%	Issue Age	Deaths	%
1,2,3	17	118	0–39	15	106
4 & 5	8	–	40–up	21	74
1 – 5	25	116	ALL	36	85
6 – 10	7	–			
11 – 25	4	–			
6 – 25	11	52			
1 – 25	36	85			

3 – 5 years from last attack

Policy Years	Deaths	%	Issue Age	Deaths	%
1,2,3	18	113	0–39	15	90
4 & 5	8	–	40–up	37	89
1 – 5	26	99	ALL	52	89
6 – 10	13	79			
11 – 25	13	83			
6 – 25	26	81			
ALL	52	89			

AFTER 5 years from last attack

Policy Years	Deaths	%	Issue Age	Deaths	%
1,2,3	9	112	0–39	7	–
4 & 5	1	–	40–up	15	65
1 – 5	10	76	ALL	22	77
6 – 10	7	–			
11 – 25	5	–			
6 – 25	12	78			
1 – 25	22	77			

ALL years after last attack

Policy Years	Deaths	%	Issue Age	Deaths	%
1,2,3	44	115	0–39	37	103
4 & 5	17	76	40–up	73	78
1 – 5	61	100	ALL	110	85
6 – 10	27	74			
11 – 25	22	68			
6 – 25	49	71			
1 – 25	110	85			

The most obvious demonstration from this display is that the excess mortality is limited to the earlier years in all tables. In the third year of all our groups combined there were only six deaths yielding a mortality of less than 50 per cent. None of these deaths was remotely related to ulcer so that in point of actual fact, all the excess mortality occurred in the first two years, the actual figures for which are 38 deaths yielding a mortality of 146 per cent.

The age incidence of the early mortality is interesting. You will note from the "all years after attack" table that the mortality at issue ages 0–39 is 103 and at ages 40 up it is 78. A further analysis by policy year follows:

Policy Year	Ages 0–39 Deaths	%	Ages 40 up Deaths	%
1 & 2	15	178	23	131
others	22	80	50	66
ALL	37	103	73	78

The concentration of mortality involves chiefly the younger ages, though it is far from absent later.

A review of the 38 deaths in these first two years is enlightening. Four were due to ulcer hemorrhage; three followed perforation and two were surgical, in both cases after partial resection. In addition to these nine, there were five instances of stomach malignancy indicating diagnostic error. As you recognize, these incidences are far above the normal and more than account for the excess mortality.

Note that the excess of the first three years does not vary materially according to the period from the last attack. Note finally that after the first two policy years the mortality at all durations is less than normal. Despite these low ultimate figures, ulcer deaths involving seven policies occurred in years 5, 9, 10, 11, 13 and 21 respectively. The causes were the usual hemorrhage and perforation. The 13th year death involved two policeis and was attributed to a curious atropine poisoning complicating ulcer treatment. There was no surgical mortality in the ultimate period. I have given this detail in order to point out the quite even distribution of these deaths and the fact that their number exceeds the normal by many times. Had deaths from ulcer followed a normal incidence, the ultimate mortality would have been about ten points lower.

The insurance picture of duodenal ulcer without operation seems to indicate that except in the first two years the absence of any operation is an indication that the disease is mild.

In the first two years there is anti-selection as to severity of the disease especially at the younger ages. There is also

some serious diagnostic error at the older ages. These two influences combine to produce a definite excess of mortality which, however, can be satisfactorily covered by a modest average rating.

These studies demonstrate the life-long character of even mild ulcer disease and in addition they show that fundamentally the ulcer constitution has a strikingly low mortality; for despite regularly distributed ulcer deaths many times the normal incidence, the ultimate mortality remains satisfactory. We attempted to demonstrate something of some of the diathetic panels. We divided our cases into two age groups and for each we constructed distribution curves for the build-weight variations from our normals. We then compared these with similar curves constructed from adequate and parallel samples of standard issues. We could detect no significant differences. Nor do the blood pressures appear to vary significantly.

I believe that the matter of diathesis deserves more attention than it has received in life insurance medicine. Diathetic factors are certainly responsible for the low mortalities of slow pulse groups despite the intrusion of some heart block cases. The same is true of functional nervous system impairment groups despite the effects of diagnostic error and the abnormal incidence of suicide. Diathesis may also be the dominant influence behind the favorable mortality of women, though environment receives more credit at present.

Much has been written and spoken concerning the environmental exciting factors responsible for ulcer. Our studies add nothing new. Incidentally, in a private communication, Dr. Tsugitake Isshiki, Medical Director of the Asahi Mutual Life of Tokyo, tells me that the incidence of ulcer deaths in the mortality of his company has increased markedly since the close of the war. The figures are a little difficult to compare but the increase appears to approach about 35 per cent. This has occurred despite a much improved food situation and increase in weight and blood pressure from the low war time levels. The doctor states he does not know why the increase is occurring.

DUODENAL ULCER—OPERATED

It is the distinction of this division that, presumably in every case, the fact of ulcer and its site have been determined with precision by direct local observation at operation. Almost invariably these critical details are recorded somewhere and are available if the effort to obtain them is deemed practical and justified. Although the group should be rather more homogeneous than is the "without operation" group, it is smaller—only a little more than a third as large. For this reason, some of the sections of the sub-groups are too small to tell a dependable story although I list them herewith:

0 – 2 years from operation

Policy Years	Deaths	%	Issue Age	Deaths	%
1,2,3	6	–	0–40	4	–
4 & 5	1	–	40–up	13	174
1 – 5	7	–	ALL	17	142
6 – 10	5	–			
11 – 25	5	–			
6 – 25	10	129			
ALL	17	142			

3 – 5 years from operation

Policy Years	Deaths	%	Issue Age	Deaths	%
1,2,3	3	–	0–40	8	–
4 & 5	3	–	40–up	22	108
1 – 5	6	–	ALL	30	111
6 – 10	12	157			
11 – 25	12	137			
6 – 25	24	146			
ALL	30	111			

AFTER 5 years from operation

Policy Years	Deaths	%	Issue Age	Deaths	%
1,2,3	8	–	0–40	2	–
4 & 5	0	–	40–up	19	112
1 – 5	8	–	ALL	21	105
6 – 10	7	–			
11 – 25	6	–			
6 – 25	13	115			
1 – 25	21	105			

ALL years after operation

Policy Years	Deaths	%	Issue Age	Deaths	%
1,2,3	17	119	0–40	14	98
4 & 5	4	–	40–up	54	121
1 – 5	21	89	ALL	68	115
6 – 10	24	143			
11 – 25	23	123			
6 – 25	47	133			
1 – 25	68	115			

It appears as a trend that the farther from operation, the better the mortality, and that the excess in the group characterized as "0–2 years from operation" is substantial; but our material is none too voluminous and the conclusion needs confirmation. In the table combining "all years since operation", the mortality of the first three years is almost exactly that of the same period among the unoperated cases. Again the third year mortality is low—probably accidentally so. In this year there were only four claims involving three lives and none of the deaths could be traced even remotely to ulcer. The concentration in the first two years is evidenced by 13 claims yielding a mortality of 137 per cent. Four of these deaths were due to ulcer—two from hemorrhage and two following operation. One of these operations was a partial resection, the other was performed for gastrojejunocolic fistula though further details are lacking. There were no cancer deaths during these early years and no evidence of fatal mistaken diagnosis.

The most important finding of this part of the study is the excess mortality experienced in both the early and late sections of the ultimate period.

Among our 6–10 years' exposures, the mortality was 143 per cent. Of the 24 claims involved, no fewer than six claims on five lives were due to ulcer. Of these, 2 occurred in the 6th year, 1 in the 7th, 2 in the 8th and 1 in the 9th. Ulcer and hemorrhage caused 2 each and in 2 the details were not given except for ulcer being the cause of death.

We experienced 23 claims in the 11–25 year group. Of these, 4 claims were due to ulcer. Three claims involving 2 lives occurred from hemorrhage in the 16th and 18th years respectively and a surgical death followed partial gastrectomy in the 22nd year. Again note the even distribution of ulcer mortality much in excess of the normal incidence.

As to the type of operation that preceded issue in the claim cases, details were inadequate in 10 claims involving 9 lives. In 49 claims on 43 lives, gastroenterostomy had been performed. In 7 deaths, various kinds of simple suture or local excision had been done. Pyloroplasty and, interestingly enough, partial gastric resection each appear in the list only once. We had no claims in cases of vagotomy, undoubtedly because its popularity is of too recent date.

In the display by issue age, the applicants who were older on admission are evidently at a disadvantage. If 13 deaths can be depended upon to tell a story, the older age mortality is concentrated in the "0–2 years from operation" group. Again the scantiness of data makes corroboration desirable.

Our study, as a whole, of operated cases indicates that, for practical purposes, the fact of operation means a more severe degree of ulcer disease and therefore at entry the status as to operation is a critical datum. In the first two or three years, the mortality is about that of unoperated cases, or perhaps I should say the mortality of unoperated cases is as bad as that of operated cases. After the select period, the deaths are increased over an indefinite period, despite the favorable basic mortality which, as I have told you, we suspect is conferred by the ulcer diathesis.

Our data gives indirect support to those who hold that gastric resection gives the best ultimate result mortality-wise, though we also can testify as to the high immediate mortality of the procedure as it is performed throughout the country. What immediate surgical mortality we have been able to recognize has been associated almost entirely with this operation. We have found no deaths from pernicious anemia in the group.

I probably should sit down at this point. My copy of the paper which I was to discuss is headed "Ulcers of the Duodenum" and I have had my say upon that subject—however, in the official program the subject is "Peptic Ulcer", and also Dr. Wilson has made frequent reference to ulcer of the stomach. I can at least give you our experience.

STOMACH ULCER—WITH OPERATION

0 – 2 years from operation

Policy Years	Deaths	%	Issue Age	Deaths	%
1,2,3	2	–	0–39	3	–
4 & 5	0	–	40–up	2	–
1 – 5	2	–	ALL	5	–
6 – 10	0	–			
11 – 25	3	–			
6 – 25	3	–			
1 – 25	5	–			

3 – 5 years from operation

Policy Years	Deaths	%	Issue Age	Deaths	%
1,2,3	5	–	0–39	6	–
4 & 5	12	541	40–up	27	203
1 – 5	17	297	ALL	33	204
6 – 10	7	–			
11 – 25	9	–			
6 – 25	16	153			
1 – 25	33	204			

AFTER 5 years from operation

Policy Years	Deaths	%	Issue Age	Deaths	%
1,2,3	10	183	0–39	10	236
4 & 5	6	–	40–up	28	118
1 – 5	16	176	ALL	38	136
6 – 10	10	142			
11 – 25	12	102			
6 – 25	22	117			
1 – 25	38	136			

ALL years from operation

Policy Years	Deaths	%	Issue Age	Deaths	%
1,2,3	17	175	0–39	19	253
4 & 5	18	297	40–up	57	149
1 – 5	35	222	ALL	76	166
6 – 10	17	150			
11 – 25	24	128			
6 – 25	41	136			
1 – 25	76	166			

Since we have included stomal ulcers here, the group represents a still futher intensity of ulcer disease which is reflected in higher early mortality. The excess of deaths is well maintained throughout life as far as we could trace it.

STOMACH ULCER—WITHOUT OPERATION

0 – 2 years from last attack

Policy Years	Deaths	%	Issue Age	Deaths	%
1,2,3	5	–	0–39	4	–
4 & 5	0	–	40–up	3	–
1 – 5	5	–	ALL	7	–
6 – 10	1	–			
11 – 25	1	–			
6 – 25	2	–			
1 – 25	7	–			

3 – 5 years from last attack

Policy Years	Deaths	%	Issue Age	Deaths	%
1,2,3	3	–	0–39	4	–
4 & 5	5	–	40–up	10	92
1 – 5	8	–	ALL	14	85
6 – 10	2	–			
11 – 25	4	–			
6 – 25	6	–			
1 – 25	14	85			

AFTER 5 years from last attack

Policy Years	Deaths	%	Issue Age	Deaths	%
1,2,3	6	–	0–39	10	179
4 & 5	4	–	40–up	15	64
1 – 5	10	100	ALL	25	87
6 – 10	5	–			
11 – 25	10	96			
6 – 25	15	79			
1 – 25	25	87			

ALL years from last attack

Policy Years	Deaths	%	Issue Age	Deaths	%
1,2,3	14	111	0–39	18	129
4 & 5	9	118	40–up	28	77
1 – 5	23	114	ALL	46	91
6 – 10	8	–			
11 – 25	15	95			
6 – 25	23	76			
1 – 25	46	91			

This is the group toward which Dr. Wilson's criticisms can be most effectively directed. I do not have any idea that this reflects the mortality of true unoperated stomach ulcer. In fact, these mortalities are so parallel to those of duodenal ulcer that I think it presents very good evidence that most of these cases *are* duodenal, and that in handling unoperated peptic ulcer cases the presumption for classification is always in favor of duodenal ulcer.

In summary, my discussion has tried to do several things:

1. To insist that mortality studies of the ulcerous insured are of value.
2. To promote the thesis that there are varying degress of severity of ulcer disease recognizable in numerous cases. The mildest are those Dr. Wilson has described as unrecognized yet occurring frequently. These are the cases that gave his company the mortality incidence of only 0.3 per cent. Next are those recognized at entry but not operated upon. The mortality of the earliest years is clearly elevated because we have not yet learned to pierce the camouflage of anti-selection nor to recognize the diagnostic errors. After those early years, the mortality is standard. Next in severity are the operated cases which give life-long increased mortality. Finally come the cases recurrent after operation, for example, the stomal ulcer which yields a high and persistent death rate.
3. That such data as number and frequency of "attacks"—vague as they may be—are data of value for classification purposes, and even "duration from operation" seems influential in the surgical cases.

I have enjoyed the study of Dr. Wilson's paper. It is erudite and original. He has added another bit of lore to the literature of this important and puzzling segment of the impairment field.

PRESIDENT ROBINSON—Dr. Wilson's paper is now open for discussion from the floor.

DR. RICHARD C. MONTGOMERY—I was very pleased to see this subject included on the agenda. I think probably if you were to ask the policy of ten medical officers here at the meeting, you would get ten different answers.

Some time ago, someone told me I had an ulcer, so I am very definitely interested in this subject. I fear that my opinions differ slightly from those in Dr. Wilson's paper. However, I hope that he is right. Some time ago, a group of our medical officers discussed this subject, having in mind

the possibility of revising our suggested ratings. After working on the subject for a year, we finally decided that compromise was the only solution. Now, I do not really know what to do—whether I should go and have a drink of milk or whether I should go and have a drink of whiskey. However, I think I should wait until I have had the opportunity of reading Dr. Wilson's paper and the discussion.

PRESIDENT ROBINSON—Is there any further discussion?

DR. DINGMAN—I have four questions and all pertain to duodenal ulcer.

1. How do you rate applicants with duodenal ulcer, who have not been disabled, have not had hemorrhage, or have not been hospitalized?

2. How do you rate duodenal ulcer applicants who have had hemorrhage or have been hospitalized?

3. How do you rate duodenal ulcer applicants who have had surgical treatment?

4. How do you differentiate in rating between gastroenterostomy and vagotomy?

PRESIDENT ROBINSON—Dr. Wilson, it seems to me that there has been great interest aroused in your paper and Dr. McAlister's discussion. Will you close the discussion?

DR. WILSON—First, I want to make some comments with regard to Dr. McAlister's figures which show a greater loss ratio during the early years. That is a subject that is bothering the actuaries. The tables I showed early in the paper revealed high incidence of death at the earlier stages, which cannot be explained. It is very large and it does not mean ulcers. It means all categories of conditions that produce death.

I might just mention that in connection with some early deaths among ulcer cases there were very few who actually died of ulcer. Two of the group were killed in automobile accidents. One death resulted from an aeroplane catastrophe. It seems to me that such deaths at the younger ages reflect the general hazards of the group.

I want to mention an investigation made some years ago, because it happens to tie in directly with our present group as regards age at death. On the average, death occurred at the age of 59, or in the sixtieth year. Some years ago, Emery and Monroe analyzed 1,435 cases at the Peter Bent Brigham Hospital, including follow-up studies, and found that the average age at death was 59 years. They concluded that in the average case the disease does not shorten longevity.

Now, to answer Dr. Dingman, we have not modified our action in conformity with the results of this study. However, we did modify our action and devise a new table for our manual about four years ago. We completely discarded the old type of table which referred to the number of attacks and the time elapsed since the last attack. The following are our new selection tables:

	ACTION	D.P.	A.I.P.
Duodenal ulcer, without operation.			
Duodenal ulcer, with operation.			
A. No history of hemorrhage — No surgical treatment.			
1. Single acute episode with active treatment, rest, diet, medication, recovery and full-time occupation has been followed for at least 6 months.			
After 6 months — careful of diet or otherwise cautious.	150%	Y*	Y*
After 1 Year — no diet, signs or symptoms.	S	Y	Y
2. History of — intermittent, acute exacerbations, possibly over several years.			
After 6 months from latest episode, with full-time occupational activities—no treatment beyond caution in eating.	150%	Y*	Y*
After 2 Years — without symptoms, medicinal treatment or restriction in activities — full diet.	S	Y	Y
3. No acute episodes — History of subacute or chronic character — No medicinal treatment — Symptoms controlled by avoiding certain irritants in diet. Never any interruption of full occupational activities.	S	Y	Y

	ACTION	D.P.	A.I.P.
4. Subacute or chronic type where, with no history of interruption of full occupational duties, there has been modification of diet and interval use of medication and/or bed-time meal.	150%	Y*	Y*
After 3 Years of no signs, symptoms, medication or modification of diet beyond elimination of certain irritant foods.	S	Y	Y
B. Cases with history of hemorrhage or of operation.			
Within 1 Year	P		
After 1 Year with full occupational duties performed with no signs or symptoms, medication or modification of diet beyond elimination of certain irritant foods.	150%	Y*	Y*
After 5 Years from latest hemorrhage or surgical procedure, with no interval signs, symptoms, medication or modification of diet beyond elimination of certain irritant foods.	S	Y	Y

N. B. (1) Limit to $50,000 except in selected cases after five or more years with freedom from signs or symptoms, no medication or modification of diet beyond elimination of certain irritant foods. Consider for retention limits.

N. B. (2) Where cases issued substandard in amounts of $25,000 or more are being considered for rating modification, require up-to-date x-ray evidence.

	ACTION	D.P.	A.I.P.
Ulcer of stomach without operation.			
Ulcer of stomach with operation.			
Consider under following tables when, through information from attendings plus data developed by examination, it is satisfactorily established that no malignancy exists or is suspected.			
(a) Treated medically or by surgical excision of ulcer			
Within 2 Years.	P	P	P
2 — 5 Years — symptom free and no treatment			
Under Age 40	137%–175%	D	D
Over Age 40	150%–225%	D	D
Over 5 Years — symptom free and no treatment	S –137%	Y–Y*	Y–Y*
Limit — $25,000 for ten years.			

	ACTION	D.P.	A.I.P.
(b) Treated by gastroenterostomy or partial gastrectomy.			
Within 1 Year	P	P	P
After 1 full year — no symptoms or treatment — weight regained and maintained			
2nd — 5th Years	150%–225%	D	D
5th and 7th Years	125%–137%	Y*	Y*
Over 7 Years	S	Y	Y
Limit to $25,000 for 10 Years — $50,000 after 10 Years.			

SYMBOLS

D Decline
S Standard
P Postpone
Y Approved
Y* 1. Considered on facts of individual case
2. Approved for limited amounts
3. Increase in rate (1½ or 2) may be required

PRESIDENT ROBINSON—It is now my very great pleasure to call on our President-elect, Dr. Edwin G. Dewis. The affairs of The Association of Life Insurance Medical Directors are in good hands. Good luck to you!

PRESIDENT-ELECT DEWIS—Thank you, gentlemen. I have no intention at all of making a speech, but I do want to thank you for this very high honor you have conferred upon me, and hope that with your assistance and co-operation we shall be able to carry on the high tradition that Dr. Robinson and his distinguished predecessors have handed down to us.

Is there any further business to come before the meeting?

DR. ROBERT L. ROWLEY—Mr. President and Members of the Association: As we reach this moment when we are about to turn our faces toward home, let us pause long enough to express our feelings about this splendid meeting to be brought to a close.

Our retiring President, Dr. Robinson, and those who have assisted him in preparing and carrying through such a program, filled as these two days have been with the unfolding of scientific information, adaptable for our use in medical

underwriting—these persons, and also those who have contributed to the discussions, deserve expressions of appreciation. In the form of a resolution, it might read as follows: that we now assembled do give formal expression of our thanks to those who have made possible this interesting and valuable meeting of our Association.

Mr. President, I should like to move the adoption of this resolution with a rising vote.

PRESIDENT-ELECT DEWIS—May we have a second? The motion is seconded. May we have a rising vote, gentlemen, on that resolution.

Gentlemen, if there is no further business, the meeting will stand adjourned.

* * *

The following doctors were present at some time during the sessions:

J. W. Abbott
C. B. Ahlefeld
H. H. Amiral
K. W. Anderson
T. M. Armstrong
W. B. Aten
J. A. Avrack

H. A. Bancel
N. J. Barker
G. P. Barnett
C. F. Beach, Jr.
J. R. Beard
E. W. Beckwith
J. E. Bee
M. F. Bell
M. B. Bender
C. C. Berwick
J. R. Biggs
C. C. Birchard
W. R. Bishop
J. E. Boland
William Bolt
J. T. Bowman
K. F. Brandon

A. W. Bromer
F. R. Brown
H. B. Brown
Leslie Brown
R. F. Buchan
E. R. Bush
B. F. Byrd

E. J. Campbell
P. E. Carlisle
D. W. Carter, Jr.
R. T. Cathcart
V. S. Caviness
W. E. Chamberlain
J. P. Chapman
P. H. Charlton
J. C. Clifford
E. E. Clovis
H. A. Cochran
N. B. Cole
B. R. Comeau
F. R. Congdon
C. A. R. Connor
J. L. Cook
F. V. Costello

H. D. Delamere
Aniceto Del Rio
P. G. Denker
E. J. Dewees
E. G. Dewis
T. H. Dickson
F. R. Dieuaide
E. S. Dillon
H. W. Dingman
J. P. Donelan
G. D. Dorman
W. W. Dow
L. B. Dunn

W. W. Eakin
T. M. Ebers
L. B. Ellis
J. L. Evans

A. H. Faber
J. G. Falconer
R. K. Farnham
H. H. Fellows
R. M. Filson
Frederick Fink

P. M. L. Forsberg
E. M. Freeland
H. M. Frost

D. S. Garner
J. H. Geddes
J. T. Geiger
W. M. Genthner
E. E. Getman
R. T. Gilchrist
R. A. Goodell
George Goodkin
H. W. Goos
A. S. Graham
Ghent Graves
H. M. Gray
G. E. Greenway
R. S. Gubner
J. R. Gudger

Llewellyn Hall
G. W. Halpenny
O. E. Hanes
A. H. Hansen
T. R. Harrison
L. E. Hathaway
H. L. Hauge
W. C. Hausheer
E. M. Henderson
E. V. Higgins
W. L. Hilliard
E. C. Hillman, Jr.
D. W. Hoare
J. C. Horan
E. G. Howe
T. B. Hoxie
J. H. Humphries
J. J. Hutchinson
J. R. B. Hutchinson

A. S. Irving
J. G. Irving

A. O. Jimenis
A. E. Johann
J. W. Johnson

E. A. Keenleyside
N. R. Kelley
E. R. Kerby
H. B. Kidd
C. E. Kiessling
D. G. Kilgore
H. B. Kirkland
Edward Kuck

P. H. Langner, Jr.
L. G. LaPointe
R. C. Larcom, Jr.
A. L. Larson
I. C. Lawler
L. H. Lee
E. P. Leeper
H. R. Leffingwell
E. H. Lindstrom
J. M. Livingston
G. J. Lunz

H. C. McAlister
F. A. McChesney
C. B. McCulloch
A. J. McGanity
F. J. McGurl
W. G. McLaughry
George McLean
L. L. McLellan
A. R. McMahan
W. J. McNamara

Charles Maertz
Ross Magee
S. J. N. Magwood
R. W. Mann
N. C. Marvel
Olan Meeker
J. T. Montgomery
R. C. Montgomery
C. V. Mulligan

S. A. Narins
R. A. Nelson
R. E. Nicholson

M. I. Olsen
B. H. Olson

W. C. Page
C. B. Parker
A. E. Parks
J. M. Peck
D. S. Pepper
G. S. Pesquera
C. A. Peters
J. C. Pierson
C. B. Piper
Cullen Pitt
J. J. Post
R. W. Pratt
M. A. Puzak

E. J. Quinn

O. S. Randall
J. H. Ready
C. L. Reeder
H. M. Rees
P. V. Reinartz
W. A. Reiter
W. M. Reynolds
G. P. Robb
D. C. Roberts
A. J. Robinson
Gordon Ross
R. L. Rowley
H. A. Rusk
Norman Ruud

K. F. Schaefer
B. T. D. Schwarz
W. H. Scoins
Albert Seaton
R. C. Secor
D. L. Selby
J. T. Sheridan
D. M. Shewbrooks
R. R. Simmons
W. A. Smith
C. G. Spivey
F. L. Springer

H. F. Starr
J. B. Steele
D. F. R. Steuart
L. Q. Stewart
I. R. Stidger
F. M. Stites
S. J. Streight
L. G. Sykes
B. C. Syverson

L. J. Tedesco
K. J. Thomson
Joseph Travenick, Jr.
F. D. Truax
Maurice Turcotte

H. E. Ungerleider

Euen Van Kleeck
A. E. Venables
R. C. Voss

Murray Wagman
F. A. Waldron
K. E. Ward
F. A. Warner
C. F. Warren
R. L. Weaver
Jefferson Weed
J. H. Weidner
D. E. W. Wenstrand

S. S. Werth
C. F. S. Whitney
H. E. Wiley
J. A. Wilhelm
E. S. Williams
R. L. Willis
A. C. Wilson
C. L. Wilson
M. C. Wilson
G. E. Woodford

L. S. Ylvisaker
A. W. Young
G. G. Young.

Also present were:

Clarence Axman

Preston Bassett

Charles Edwards

George Greening

E. J. Hardin
Stephen Hodges
Arthur Hunter

E. A. Lew
Miss A. M. Lyle

Brien McMahon

Herbert Marks
John Mills

E. M. Neumann

G. A. Oliver

H. M. Parker
L. N. Parker

Francis Reilly
Edward Ruge

Pearce Shepherd

John Thatcher
A. E. Thyselius

P. V. Wells
H. E. White
J. C. Wilberding
F. B. Wilde

Total attendance at all sessions, 261.

In Memoriam

Deceased Since Fifty-sixth Annual Meeting

William M. Bradshaw, M. D.
Mutual Life Insurance Company of New York
Died May 1, 1948

Frank H. Carber, M. D.
Mutual Life Insurance Company of New York
Died February 28, 1948

Herschel F. Connally, M. D.
Amicable Life Insurance Company
Died July 16, 1948

William D. Heaton, M. D.
New York Life Insurance Company
Died January 14, 1948

Augustus S. Knight, M. D.
Metropolitan Life Insurance Company
Died March 21, 1948

Henry B. Turner, M. D.
Guardian Life Insurance Company
Died October 5, 1948

DECEASED MEMBERS

John L. Adams, M. D.	New York, N. Y.
Charles D. Alton, M. D.	Hartford, Conn.
Malcolm O. Austin, M. D.	San Francisco, Calif.
Walter C. Bailey, M. D.	Boston, Mass.
Henry A. Baker, M. D.	Kansas City, Mo.
A. W. Barrows, M. D.	Hartford, Conn.
John T. J. Battle, M. D.	Greensboro, N. C.
Wesley W. Beckett, M. D.	Los Angeles, Calif.
Charles D. Bennett, M. D.	Newark, N. J.
Emmanuel P. Benoit, M. D.	Montreal, Canada
Charles Bernacki, M. D.	New York, N. Y.
Thomas W. Bickerton, M. D.	New York, N. Y.
Albert W. Billing, M. D.	New York, N. Y.
Wilton F. Blackford, M. D.	Louisville, Ky.
David N. Blakely, M. D.	Boston, Mass.
Robert J. Blanchard, M. D.	Winnipeg, Canada
Harold E. Bogart, M. D.	New York, N. Y.
William M. Bradshaw, M. D.	New York, N. Y.
Frederick G. Brathwaite, M. D.	New York, N. Y.
William R. Bross, M. D.	New York, N. Y.
Chauncey R. Burr, M. D.	New York, N. Y.
Robert L. Burrage, M. D.	Newark, N. J.
James Campbell, M. D.	Hartford, Conn.
Frank H. Carber, M. D.	New York, N. Y.
Willard B. Carpenter, M. D.	Columbus, Ohio
Frank W. Chapin, M. D.	New York, N. Y.
Frederick W. Chapin, M. D.	Springfield, Mass.
Ferdinand E. Chatard, M. D.	Baltimore, Md.
Charles L. Christiernin, M. D.	New York, N. Y.
Henry Colt, M. D.	Pittsfield, Mass.
Herschel F. Connally, Sr., M. D.	Waco, Texas
Henry W. Cook, M. D.	Minneapolis, Minn.
Donald B. Cragin, M. D.	Hartford, Conn.

DECEASED MEMBERS

Thomas C. Craig, M. D.	New York, N. Y.
Hugh W. Crawford, M. D.	Boston, Mass.
Hamilton C. Cruikshank, M. D.	Toronto, Canada
Edward Curtis, M. D.	New York, N. Y.
Clark W. Davis, M. D.	Cincinnati, Ohio
William B. Davis, M. D.	Cincinnati, Ohio
Charles A. Devendorf, M. D.	Detroit, Mich.
Henry K. Dillard, M. D.	Philadelphia, Pa.
Frank Donaldson, M. D.	Baltimore, Md.
Percy G. Drake, M. D.	Hartford, Conn.
Edwin W. Dwight, M. D.	Boston, Mass.
James B. Eagleson, M. D.	Seattle, Wash.
Z. Taylor Emery, M. D.	New York, N. Y.
Joseph E. Engelson, M. D.	New York, N. Y.
Francis C. Evers, M. D.	New York, N. Y.
William G. Exton, M. D.	Newark, N. J.
Robert H. Feldt, M. D.	Milwaukee, Wis.
John W. Fisher, M. D.	Milwaukee, Wis.
Paul FitzGerald, M. D.	Newark, N. J.
Thomas A. Foster, M. D.	Portland, Me.
Robert A. Fraser, M. D.	New York, N. Y.
Samuel W. Gadd, M. D.	Philadelphia, Pa.
Homer Gage, M. D.	Worcester, Mass.
Thomas H. Gage, M. D.	Worcester, Mass.
Donald M. Gedge, M. D.	New York, N. Y.
Walter R. Gillette, M. D.	New York, N. Y.
Frank S. Grant, M. D.	New York, N. Y.
Frederick L. Grasett, M. D.	Toronto, Canada
Landon Carter Gray, M. D.	New York, N. Y.
Frederick W. Hagney, M. D.	Newark, N. J.
Ignatius Haines, M. D.	Boston, Mass.
George C. Hall, M. D.	Hartford, Conn.
Joseph B. Hall, M. D.	Newark, N. J.
Edward H. Hamill, M. D.	Richmond, Va.

DECEASED MEMBERS

William D. Heaton, M. D.	New York, N. Y.
William W. Hitchcock, M. D.	Los Angeles, Calif.
Angier B. Hobbs, M. D.	New York, N. Y.
William W. Hobson, M. D.	Pittsburgh, Pa.
Donald C. Hoffman, M. D.	New York, N. Y.
Edgar Holden, M. D.	Newark, N. J.
John Homans, M. D.	Boston, Mass.
John Homans, 2d, M. D.	Boston, Mass.
J. Charles Humphreys, M. D.	Philadelphia, Pa.
Abel Huntington, M. D.	New York, N. Y.
Ross Huston, M. D.	Des Moines, Iowa
Henry H. Hutchison, M. D.	Toronto, Canada
Lefferts Hutton, M. D.	New York, N. Y.
Charles E. Iliff, M. D.	Cincinnati, Ohio
Phineas H. Ingalls, M. D.	Hartford, Conn.
Charles B. Irwin, M. D.	Chicago, Ill.
Arthur Jukes Johnson, M. D.	Toronto, Canada
William M. Jones, M. D.	Greensboro, N. C.
John M. Keating, M. D.	Philadelphia, Pa.
Edward B. Kellogg, M. D.	Boston, Mass.
Frank W. Kenney, M. D.	Denver, Colo.
Augustus S. Knight, M. D.	New York, N. Y.
William W. Knight, M. D.	Hartford, Conn.
Edward Lambert, M. D.	New York, N. Y.
John B. Lewis, M. D.	Hartford, Conn.
Ernest H. Lines, M. D.	New York, N. Y.
John M. Little, M. D.	Boston, Mass.
Robert L. Lounsberry, M. D.	Binghamton, N. Y.
Carl Lovelace, M. D.	Waco, Texas
Henry P. Lyster, M. D.	Detroit, Mich.
Lewis F. MacKenzie, M. D.	Newark, N. J.
Milton T. McCarty, M. D.	Frankfort, Ind.
Charles N. McCloud, M. D.	St. Paul, Minn.
Francis A. McGreen, M. D.	New York, N. Y.

DECEASED MEMBERS

Lewis McKnight, M. D.	Milwaukee, Wis.
Thomas H. McMahon, M. D.	Toronto, Canada
Walter T. McNaughton, M. D.	Milwaukee, Wis.
William L. Mann, M. D.	Winnipeg, Canada
Elias J. Marsh, M. D.	Paterson, N. J.
Henry A. Martelle, M. D.	Hartford, Conn.
Edward L. Mathias, M. D.	Kansas City, Mo.
Allison Maxwell, M. D.	Indianapolis, Ind.
Archibald Mercer, M. D.	Newark, N. J.
Francis D. Merchant, M. D.	New York, N. Y.
William R. Miller, M. D.	Hartford, Conn.
William D. Morgan, M. D.	Hartford, Conn.
John P. Munn, M. D.	New York, N. Y.
William Natress, M. D.	Toronto, Canada
Charles T. Necker, M. D.	Waterloo, Canada
Edwin M. Northcott, M. D.	Portland, Me.
Ralph B. Ober, M. D.	Springfield, Mass.
Brace W. Paddock, M. D.	Pittsfield, Mass.
Frank K. Paddock, M. D.	Pittsfield, Mass.
J. Allen Patton, M. D.	Newark, N. J.
William O. Pauli, M. D.	Cincinnati, Ohio
William A. Peterson, M. D.	Chicago, Ill.
Joseph E. Pollard, M. D.	Newark, N. J.
William E. Porter, M. D.	New York, N. Y.
Albert T. Post, M. D.	New York, N. Y.
James T. Priestly, M. D.	Des Moines, Iowa
William W. Quinlan, M. D.	Chicago, Ill.
Oliver P. Rex, M. D.	Philadelphia, Pa.
James A. Roberts, M. D.	Toronto, Canada
Thomas H. Rockwell, M. D.	New York, N. Y.
Oscar H. Rogers, M. D.	New York, N. Y.
Edward K. Root, M. D.	Hartford, Conn.
James F. W. Ross, M. D.	Toronto, Canada
Charles L. Rudasill, M. D.	Richmond, Va.

DECEASED MEMBERS

Gurdon W. Russell, M. D.	Hartford, Conn.
Robert Sanderson, M. D.	Boston, Mass.
Joseph O. Segura, M. D.	Jackson, Miss.
George R. Shepherd, M. D.	Hartford, Conn.
Arthur L. Sherrill, M. D.	New York, N. Y.
Donald W. Skeel, M. D.	Los Angeles, Calif.
Dewitt Smith, M. D.	Dallas, Tex.
George S. Stebbins, M. D.	Springfield, Mass.
Archibald R. Stone, M. D.	Columbus, Ohio
George S. Strathy, M. D.	Toronto, Canada
Melancthon Storrs, M. D.	Hartford, Conn.
Brandreth Symonds, M. D.	New York, N. Y.
H. Cabell Tabb, M. D.	Richmond, Va.
Harold F. Taylor, M. D.	Hartford, Conn.
James Thorburn, M. D.	Toronto, Canada
James D. Thorburn, M. D.	Toronto, Canada
William Thorndike, M. D.	Milwaukee, Wis.
Paul E. Tiemann, M. D.	New York, N. Y.
Harry Toulmin, M. D.	Philadelphia, Pa.
Frank L. Truitt, M. D.	Indianapolis, Ind.
Henry Tuck, M. D.	New York, N. Y.
Henry B. Turner, M. D.	New York, N. Y.
John S. Turner, M. D.	Dallas, Tex.
Joseph P. Turner, M. D.	Greensboro, N. C.
S. Oakley Van der Poel, M. D.	New York, N. Y.
Charles A. VanDervoort, M. D.	Philadelphia, Pa.
A. L. Vanderwater, M. D.	New York, N. Y.
Clinton D. W. VanDyck, M. D.	New York, N. Y.
George A. VanWagenen, M. D.	Newark, N. J.
Aaron C. Ward, M. D.	Newark, N. J.
William Perry Watson, M. D.	Newark, N. J.
Joseph H. Webb, M. D.	Waterloo, Canada
William E. H. Wehner, M. D.	Philadelphia, Pa.
Faneuil S. Weisse, M. D.	New York, N. Y.

DECEASED MEMBERS

George R. Welch, M. D.	New York, N. Y.
Ernest A. Wells, M. D.	Hartford, Conn.
Frank Wells, M. D.	Boston, Mass.
Franklin C. Wells, M. D.	New York, N. Y.
George W. Wells, M. D.	New York, N. Y.
Charles D. Wheeler, M. D.	Worcester, Mass.
A. H. Whitridge, M. D.	Baltimore, Md.
George Wilkins, M. D.	Montreal, Canada
Thomas H. Willard, M. D.	New York, N. Y.
Charles H. Willits, M. D.	Philadelphia, Pa.
Gordon Wilson, M. D.	Baltimore, Md.
G. S. Winston, M. D.	New York, N. Y.
Harry P. Woley, M. D.	New York, N. Y.
Albert Wood, M. D.	Worcester, Mass.
Green V. Woolen, M. D.	Indianapolis, Ind.
John C. Young, M. D.	Detroit, Mich.
Joseph C. Young, M. D.	Newark, N. J.

LIST OF MEMBERS OF THE ASSOCIATION OF LIFE INSURANCE MEDICAL DIRECTORS

John W. Abbott, M. D.	Paul Revere, Worcester, Mass.
Charles B. Ahlefeld, M. D.	Business Men's, Kansas City, Mo.
George E. Allen, M. D.	National, Montpelier, Vt.
Hiram H. Amiral, M. D.	State Mutual, Worcester, Mass.
Henry H. Amsden, M. D.	United Life and Accident, Concord, N. H.
E. A. Anderson, M. D.	Modern Woodmen, Rock Island, Ill.
Karl W. Anderson, M. D.	Northwestern National, Minneapolis, Minn.
Perry A. Anderson, M. D.	Rockford Life, Rockford, Ill.
Thomas M. Armstrong, M. D.	Philadelphia Life, Philadelphia, Pa.
William B. Aten, M. D.	Security Mutual, Binghamton, N. Y.
Donald R. Auten, M. D.	New York Life, New York City
J. Albert Avrack, M. D.	United States Life, New York City
Bernard Baillargeon, M. D.	Alliance Nationale, Montreal, Canada
Henry A. Bancel, M. D.	Mutual, New York City
G. Holbrook Barber, M. D.	Manhattan, New York City
Norman J. Barker, M. D.	Connecticut General, Hartford, Conn.

Gordon P. Barnett, M. D.	Kansas City Life, Kansas City, Mo.
Charles M. Barrett, M. D.	Western and Southern, Cincinnati, Ohio
Daniel S. Baughman, M. D.	Security Life and Accident, Denver, Colo.
Carroll C. Beach, Jr., M. D.	Connecticut General, Hartford, Conn.
J. Randolph Beard, M. D.	Mutual Benefit, Newark, N. J.
Edgar W. Beckwith, M. D.	Equitable Life Assurance, New York City
James E. Bee, M. D.	Kansas City Life, Kansas City, Mo.
Roland A. Behrman, M. D.	John Hancock Mutual, Boston, Mass.
J. V. Bell, M. D.	National Fidelity, Kansas City, Mo.
Murray F. Bell, M. D.	New York Life, New York City
Maurice B. Bender, M. D.	Guardian, New York City
Roy W. Benton, M. D.	Northwestern Mutual, Milwaukee, Wis.
C. Coleman Berwick, M. D.	Metropolitan, New York City
Francis P. Bicknell, M. D.	State Mutual, Worcester, Mass.
J. Rozier Biggs, M. D.	Peoples, Washington, D. C.
Cecil C. Birchard, M. D.	Sun, Montreal, Canada
B. Cosby Bird, M. D.	Preferred, Montgomery, Ala.
William R. Bishop, M. D.	Jefferson Standard, Greensboro, N. C.
Norman R. Blatherwick, M. D.	Metropolitan, New York City
John E. Boland, M. D.	Country, Chicago, Ill.
William Bolt, M. D.	New York Life, New York City
Earl C. Bonnett, M. D.	Metropolitan, New York City
J. Thornley Bowman, M. D.	London Life, London, Canada

William E. Branch, M. D.	Constitution, Los Angeles, Calif.
Kenneth F. Brandon, M. D.	Aetna, Hartford, Conn.
Albert W. Bromer, M. D.	Metropolitan, New York City
C. Frank Brown, M. D.	Southwestern, Dallas, Texas
Frederick R. Brown, M. D.	New England Mutual, Boston, Mass.
Howard B. Brown, M. D.	Massachusetts Mutual, Springfield, Mass.
Leslie Brown, M. D.	Equitable Life Assurance, New York City
William Brueggemann, M. D.	Union Central, Cincinnati, Ohio
Ronald F. Buchan, M. D.	Prudential, Newark, N. J.
Earl R. Bush, M. D.	Western and Southern, Cincinnati, Ohio
Benjamin F. Byrd, M. D.	National Life & Accident, Nashville, Tenn.
Joseph T. Cabaniss, M. D.	Travelers, Hartford, Conn.
Edward J. Campbell, M. D.	New York Life, New York City
Hugh B. Campbell, M. D.	Phoenix Mutual, Hartford, Conn.
Paul E. Carlisle, M. D.	Prudential, Los Angeles, Calif.
David W. Carter, Jr., M. D.	Reserve Loan, Dallas, Texas
Verne S. Caviness, M. D.	Occidental, Raleigh, N. C.
Laurence D. Chapin, M. D.	Massachusetts Mutual, Springfield, Mass.
John P. Chapman, M. D.	Girard, Philadelphia, Pa.
Paul H. Charlton, M. D.	Midland Mutual, Columbus, Ohio
Edmund D. Chesebro, M. D.	Puritan, Providence, R. I.

Harry E. Christensen, M. D.	Union Mutual, Portland, Maine
Joseph C. Clifford, M. D.	Aetna, Hartford, Conn.
Milton H. Clifford, M. D.	New England Mutual, Boston, Mass.
Elijah E. Clovis, M. D.	Conservative, Wheeling, W. Va.
Augustus D. Cloyd, M. D.	Woodmen of the World, Omaha, Neb.
Harry A. Cochran, Jr., M. D.	Reliance, Pittsburgh, Pa.
Norman B. Cole, M. D.	Baltimore Life, Baltimore, Md.
Irwin E. Colgin, M. D.	Texas Life, Waco, Texas
Harry L. Colombo, M. D.	National Life, Montpelier, Vt.
Frederick R. Congdon, M. D.	Berkshire, Pittsfield, Mass.
J. Lindsay Cook, M. D.	Pilot, Greensboro, N. C.
Francis V. Costello, M. D.	Mutual, New York City
Neil L. Criss, M. D.	United Benefit, Omaha, Neb.
Howard K. Crutcher, M. D.	United Fidelity, Dallas, Texas
John P. Davis, M. D.	Security Life & Trust, Winston-Salem, N. C.
John S. Delahaye, M. D.	Empire Life, Kingston, Canada
Harold D. Delamere, M. D.	Crown, Toronto, Canada
J. Emile Desrochers, M. D.	La Sauvegarde, Montreal, Canada
Ernest J. Dewees, M. D.	Provident Mutual, Philadelphia, Pa.
Earle T. Dewey, M. D.	Metropolitan, New York City
Edwin G. Dewis, M. D.	Prudential, Newark, N. J.

Thomas H. Dickson, M. D.	Minnesota Mutual, St. Paul, Minn.
Edward S. Dillon, M. D.	Penn Mutual, Philadelphia, Pa.
Harold W. Dingman, M. D.	Continental Assurance, Chicago, Ill.
Nathaniel P. Doak, M. D.	Great Southern, Houston, Texas
Albert H. Domm, M. D.	Prudential, Los Angeles, Calif.
James P. Donelan, M. D.	Guarantee Mutual, Omaha, Neb.
Gerald D. Dorman, M. D.	New York Life, New York City
William W. Dow, M. D.	Toronto Mutual, Toronto, Canada
James T. Downs, Jr., M. D.	Fidelity Union, Dallas, Texas
Thomas C. Dunlop, M. D.	Manufacturers, Toronto, Canada
Louis B. Dunn, M. D.	Postal, New York City
John T. Eads, M. D.	Penn Mutual, Philadelphia, Pa.
William W. Eakin, M. D.	Standard, Montreal, Canada
Theodore M. Ebers, M. D.	Connecticut Mutual, Hartford, Conn.
H. Glenn Ebersole, M. D.	Illinois Bankers, Monmouth, Ill.
Laurence B. Ellis, M. D.	Boston Mutual, Boston, Mass.
Jack A. End, M. D.	Northwestern Mutual, Milwaukee, Wis.
John A. Evans, M. D.	Metropolitan, New York City
John L. Evans, M. D.	Farmers & Bankers, Wichita, Kan.
Albert H. Faber, M. D.	New York Life, New York City
J. Gilbert Falconer, M. D.	North American, Toronto, Canada

Raymond K. Farnham, M. D.	Metropolitan, New York City
Haynes H. Fellows, M. D.	Metropolitan, New York City
W. Ewart Ferguson, M. D.	Excelsior, Toronto, Canada
William S. Fewell, M. D.	Liberty, Greenville, S. C.
Ralph M. Filson, M. D.	Travelers, Hartford, Conn.
Rexford W. Finegan, M. D.	Metropolitan, New York City
Victor J. Fingar, M. D.	Equitable Life Assurance, New York City
Frederick Fink, M. D.	Franklin, Springfield, Ill.
Harry E. Flansburg, M. D.	Bankers, Lincoln, Neb.
Philip M. L. Forsberg, M. D.	United Life and Accident, Concord, N. H.
Garth E. Fort, M. D.	National Life & Accident, Nashville, Tenn.
John M. Foster, M. D.	Capitol, Denver, Colo.
Edward M. Freeland, M. D.	New York Life, New York City
Dale G. Friend, M. D.	John Hancock Mutual, Boston, Mass.
Harold M. Frost, M. D.	New England Mutual, Boston, Mass.
F. Irving Ganot, M. D.	Prudential, Newark, N. J.
I. Kenneth Gardner, M. D.	Reliance, Pittsburgh, Pa.
David S. Garner, M. D.	Shenandoah, Roanoke, Va.
J. H. Geddes, M. D.	Northern, London, Canada
John T. Geiger, M. D.	Metropolitan, New York City
William M. Genthner, M. D.	Continental American, Wilmington, Del.
Edson E. Getman, M. D.	New York Life, New York City

John M. Gilchrist, M. D.	Monarch, Springfield, Mass.
Ralph T. Gilchrist, M. D.	Northwestern Mutual, Milwaukee, Wis.
Edgar G. Givhan, Jr., M. D.	Protective, Birmingham, Ala.
Robert A. Goodell, M. D.	Phoenix Mutual, Hartford, Conn.
George Goodkin, M. D.	Equitable Life Assurance, New York City
Harry W. Goos, M. D.	Home, Philadelphia, Pa.
J. Keith Gordon, M. D.	Sun, Montreal, Canada
Angus S. Graham, M. D.	London Life, London, Canada
George M. Graham, M. D.	Lincoln National, Fort Wayne, Ind.
LeRoy C. Grau, M. D.	Travelers, Hartford, Conn.
Ghent Graves, M. D.	American General, Houston, Texas
Marvin L. Graves, M. D.	American General, Houston, Texas
Robert J. Graves, M. D.	United Life and Accident, Concord, N. H.
Harris M. Gray, M. D.	Manufacturers, Toronto, Canada
Floyd M. Green, M. D.	Columbus Mutual, Columbus, Ohio.
George E. Greenway, M. D.	Western Life Assurance, Hamilton, Canada
Frederick O. Gronvold, M. D.	Pioneer Mutual, Fargo, N. D.
Richard S. Gubner, M. D.	Equitable Life Assurance, New York City
James R. Gudger, M. D.	Mutual, New York City
Lester E. Haentzschel, M. D.	Massachusetts Mutual, Springfield, Mass.
David Halbersleben, M. D.	John Hancock Mutual, Boston, Mass.
Llewellyn Hall, M. D.	Phoenix Mutual, Hartford, Conn.

F. Tulley Hallam, M. D. State, Indianapolis, Ind.

Gerald W. Halpenny, M. D. Royal, Montreal, Canada

Ottis E. Hanes, M. D. Life Ins. Co. of Ga., Atlanta, Ga.

Charles P. Hardwicke, M. D. Western Reserve, Austin, Texas

Frank Harnden, M. D. Berkshire, Pittsfield, Mass.

Garland M. Harwood, M. D. Life Insurance Co. of Virginia, Richmond, Va.

Louis E. Hathaway, Jr., M. D. Monarch, Springfield, Mass.

Howard L. Hauge, M. D. New York Life, New York City

Walter C. Hausheer, M. D. Prudential, Newark, N. J.

Harry M. Hawkins, M. D. Old Line, Milwaukee, Wis.

Thomas L. Hawkins, M. D. Western, Helena, Mont.

Joseph K. P. Hawks, M. D. State Farm, Bloomington, Ill.

J. Harry Hayes, M. D. Union, Little Rock, Ark.

Ernest M. Henderson, M. D. Confederation, Toronto, Canada

Olin C. Hendrix, M. D. New England Mutual, Boston, Mass.

Charles R. Henry, M. D. Provident Life and Accident Chattanooga, Tenn.

Andrew C. Henske, M. D. Mutual Savings, St. Louis, Mo.

Ivan C. Heron, M. D. West Coast, San Francisco, Calif.

Eugene V. Higgins, M. D. Manhattan, New York City

William L. Hilliard, M. D. Equitable, Waterloo, Canada

Ernest C. Hillman, Jr., M. D. Mutual Benefit, Newark, N. J.

Daniel W. Hoare, M. D. Penn Mutual, Philadelphia, Pa.

Ira E. Hoffman, M. D.	Washington National, Evanston, Ill.
Joseph C. Horan, M. D.	Metropolitan, New York City
Edward G. Howe, M. D.	Prudential, Newark, N. J.
Thomas B. Hoxie, M. D.	New York Life, New York City
Henry W. Hudson, M. D.	Loyal Protective, Boston, Mass.
John L. Humphreys, M. D.	Reliance, Pittsburgh, Pa.
James H. Humphries, M. D.	Home, New York City
J. Edward Hunsinger, M. D.	Alliance, Chicago, Ill.
Benjamin L. Huntington, M. D.	John Hancock Mutual, Boston, Mass.
Samuel W. Hurdle, M. D.	Security Life & Trust, Winston-Salem, N. C.
John J. Hutchinson, M. D.	New York Life, New York City
J. Raymond B. Hutchinson, M. D.	Acacia Mutual, Washington, D. C.
William G. Hyde, M. D.	Northwestern Mutual, Milwaukee, Wis.
Albert S. Irving, M. D.	Commonwealth, Louisville, Ky.
J. Grant Irving, M. D.	Aetna, Hartford, Conn.
Samuel Jagoda, M. D.	State Reserve, Fort Worth, Texas
Albert O. Jimenis, M. D.	Metropolitan, New York City
Albert E. Johann, M. D.	Bankers, Des Moines, Iowa
Hubert R. John, M. D.	Maccabees, Detroit, Mich.
Joseph W. Johnson, Jr., M. D.	Interstate Life and Accident, Chattanooga, Tenn.
Edward A. Keenleyside, M. D.	Connecticut General, Hartford, Conn.

Charles H. Kelley, M. D.	Columbian National, Boston, Mass.
Newell R. Kelley, M. D.	Phoenix Mutual, Hartford, Conn.
Herbert B. Kennedy, M. D.	Woodmen of the World, Omaha, Neb.
Harry B. Kidd, M. D.	Metropolitan, New York City
Charles E. Kiessling, M. D.	Prudential, Newark, N. J.
Donald G. Kilgore, M. D.	Republic National, Dallas, Texas
Ray E. King, M. D.	Bankers, Des Moines, Iowa
Henry B. Kirkland, M. D.	Prudential, Newark, N. J.
Edward Kuck, M. D.	Union Central, Cincinnati, Ohio
Earl J. Kuenster, M. D.	Paul Revere, Worcester, Mass.
Paul Kurzweg, Jr., M. D.	All American Assurance, Lafayette, La.
Paul H. Langner, Jr., M. D.	Provident Mutual, Philadelphia, Pa.
Anthony J. Lanza, M. D.	Metropolitan, New York City
Louis G. LaPointe, M. D.	Equitable Life Assurance, New York City
H. Franklyn Laramore, M. D.	Connecticut Mutual, Hartford, Conn.
Rodney C. Larcom, Jr., M. D.	John Hancock Mutual, Boston, Mass.
Albert L. Larson, M. D.	Travelers, Hartford, Conn.
Ivan C. Lawler, M. D.	New York Life, New York City
Linford H. Lee, M. D.	Pacific Mutual, Los Angeles, Calif.
Edward P. Leeper, M. D.	Praetorians, Dallas, Texas
Harold R. Leffingwell, M. D.	Paul Revere, Worcester, Mass.
T. Herbert Lewis, M. D.	Western States, Fargo, N. D.

George G. Lindsay, M. D. Scranton Life, Scranton, Pa.

Everett H. Lindstrom, M. D. Western, Helena, Mont.

James A. Livingston, M. D. Liberty National, Birmingham, Ala.

John M. Livingston, M. D. Mutual, Waterloo, Canada

G. Carroll Lockard, M. D. Maryland Life, Baltimore, Md.

Cabot Lull, M. D. American, Birmingham, Ala.

Gerald J. Lunz, M. D. Knights of Columbus, New Haven, Conn.

H. Clive McAlister, M. D. Lincoln National, Ft. Wayne, Ind.

Frank M. McChesney, M. D. Equitable, Washington, D. C.

George McCreight, M. D. Bankers, Des Moines, Iowa

Carleton B. McCulloch, M. D. State, Indianapolis, Ind.

John G. MacDougall, M. D. Maritime, Halifax, Canada

Arthur J. McGanity, M. D. Dominion, Waterloo, Canada

Frank J. McGurl, M. D. Prudential, Newark, N. J.

William G. McLaughry, M. D. Protected Home Circle, Sharon, Pa.

George McLean, M. D. Sun, Baltimore, Md.

Lawrence L. McLellan, M. D. Provident Mutual, Philadelphia, Pa.

Ralph E. McLochlin, M. D. National Old Line, Little Rock, Ark.

Asher R. McMahan, M. D. Columbian Mutual, Memphis, Tenn.

William J. McNamara, M. D. Equitable Life Assurance, New York City

Charles Maertz, M. D. Union Central, Cincinnati, Ohio

Charles D. Magee, M. D.	Missouri Insurance Company, St. Louis, Mo.
S. J. Newton Magwood, M. D.	Continental, Toronto, Canada
Robert W. Mann, M. D.	Imperial, Toronto, Canada
Francis A. L. Mathewson, M. D.	Monarch, Winnipeg, Canada
Loren K. Meredith, M. D.	National, Des Moines, Iowa
Ernest B. Milan, M. D.	Peninsular, Jacksonville, Fla.
Lloyd C. Miller, M. D.	National Life & Accident, Nashville, Tenn.
Eugene Montgomery, M. D.	North American, Toronto, Canada
James T. Montgomery, M. D.	North American Reassurance, New York City
Richard C. Montgomery, M. D.	Manufacturers, Toronto, Canada
John F. Moore, Jr., M. D.	Mutual, New York City
Reuben A. Moser, M. D.	American Reserve, Omaha Neb.
Bernard Mount, M. D.	All States, Montgomery, Alabama
Elmer B. Mountain, M. D.	American Mutual, Des Moines, Iowa
Clifford V. Mulligan, M. D.	T. Eaton, Toronto, Canada
Frederick D. Munroe, M. D.	Fidelity, Regina, Canada
Sidney A. Narins, M. D.	Mutual, New York City
Richard A. Nelson, M. D.	Prudential, Newark, N. J.
Charles F. Nichols, M. D.	Penn Mutual, Philadelphia, Pa.
John B. Nichols, M. D.	Acacia Mutual, Washington, D. C.
Richard E. Nicholson, M. D.	Connecticut Mutual, Hartford, Conn.
E. Clark Noble, M. D.	National, Toronto, Canada

Andrew J. Oberlander, M. D.	National, Montpelier, Vt.
Martin J. O'Brien, M. D.	United States Life, New York City
William L. O'Connell, M. D.	Union Labor, New York City
Martin I. Olsen, M. D.	Central, Des Moines, Iowa
Baldur H. Olson, M. D.	Great-West, Winnipeg, Canada
William F. H. O'Neill, M. D.	Great-West, Winnipeg, Canada
Wilbert C. Page, M. D.	Prudential, Newark, N. J.
Charles B. Parker, M. D.	Independent Order of Foresters, Toronto, Canada
Arthur E. Parks, M. D.	Canada Life, Toronto, Canada
D. Sergeant Pepper, M. D.	Provident Mutual, Philadephia, Pa.
Gilberto S. Pesquera, M. D.	Metropolitan, New York City
Charles A. Peters, M. D.	Prudential Assurance, Montreal, Canada
Cullen Pitt, M. D.	Atlantic, Richmond, Va.
John J. Post, M. D.	Life and Casualty, Nashville, Tenn.
Roscoe W. Pratt, M. D.	New York Life, New York City
William O. Purdy, M. D.	Equitable, Des Moines, Iowa
Louis A. Pyle, M. D.	Colonial, Jersey City, N. J.
Edwin J. Quinn, M. D.	Mutual, New York City
O. Samuel Randall, M. D.	Midland National, Watertown, S. D.
Paul M. Rattan, M. D.	Great National, Dallas, Texas
James H. Ready, M. D.	General American, St. Louis, Mo.
Rezin Reagan, M. D.	Policyholder's National, Sioux Falls, S. D.

Clifton L. Reeder, M. D.	Continental Assurance, Chicago, Ill.
H. Maynard Rees, M. D.	John Hancock Mutual, Boston, Mass.
Paul V. Reinartz, M. D.	Prudential, Newark, N. J.
Walter A. Reiter, M. D.	Mutual Benefit, Newark, N. J.
Whitman M. Reynolds, M. D.	Equitable Life Assurance, New York City
Wallace R. Richardson, M. D.	National Equity, Little Rock, Ark.
H. Guy Riche, M. D.	Guaranty Income, Baton Rouge, La.
Donald F. Rikkers, M. D.	Northwestern Mutual, Milwaukee, Wis.
George P. Robb, M. D.	Metropolitan, New York City
David C. Roberts, M. D.	Guardian, New York City
Albert J. Robinson, M. D.	Connecticut General, Hartford, Conn.
Claude A. Robison, M. D.	Peoples, Frankfort, Ind.
Henry B. Rollins, M. D.	Connecticut Mutual, Hartford, Conn.
Fred W. Rolph, M. D.	Confederation, Toronto, Canada
Gordon Ross, M. D.	Massachusetts Mutual, Springfield, Mass.
Thomas F. Ross, M. D.	Ohio State, Columbus, Ohio
Edward W. Rowe, M. D.	Midwest, Lincoln, Neb.
James M. H. Rowland, M. D.	Baltimore Life, Baltimore, Md.
Robert L. Rowley, M. D.	Phoenix Mutual, Hartford, Conn.
William W. Rucks, M. D.	Home State, Oklahoma City, Okla.
Dan Y. Sage, M. D.	Southern, Atlanta, Ga.
Joe H. Sanderlin, M. D.	Pyramid, Little Rock, Ark.

Raymond C. Scannell, M. D.	Security Life and Accident, Denver, Colo.
Kenneth F. Schaefer, M. D.	Prudential, Newark, N. J.
Berthold T. D. Schwarz, M. D.	Bankers National, Montclair, N. J.
William H. Scoins, M. D.	Lincoln National, Ft. Wayne, Ind.
Ernest W. Scott, M. D.	Equitable Life Assurance, New York City.
Robert J. Scott, M. D.	Michigan Life, Detroit, Mich.
Albert Seaton, M. D.	American United, Indianapolis, Ind.
Ralph C. Secor, M. D.	Connecticut Mutual, Hartford, Conn.
David L. Selby, M. D.	Imperial, Toronto, Canada
Thomas S. Sexton, M. D.	Massachusetts Mutual, Springfield, Mass.
Hall Shannon, M. D.	Southland, Dallas, Texas
Elroy F. Sheldon, M. D.	Occidental, Los Angeles, Calif.
Joyce T. Sheridan, M. D.	Fidelity Mutual, Philadephia, Pa.
Daniel M. Shewbrooks, M. D.	Penn Mutual, Philadelphia, Pa.
Hubert H. Shook, M. D.	Ohio National, Cincinnati, Ohio
Ralph R. Simmons, M. D.	Equitable, Des Moines, Iowa
F. Hartley Smith, M. D.	Great-West, Winnipeg, Canada
Wilbur A. Smith, M. D.	Equitable Life Assurance, New York City
Morton Snow, M. D.	Massachusetts Mutual, Springfield, Mass.
Isaac Sossnitz, M. D.	Eastern, New York City
Marion Souchon, M. D.	Pan-American, New Orleans, La.
Charles G. Spivey, M. D.	Carolina Life, Columbia, S. C.
Frank L. Springer, M. D.	Columbian National, Boston, Mass.

H. Frank Starr, M. D. Jefferson Standard, Greensboro, N. C.

George G. Stebbins, M. D. Wisconsin Life, Madison, Wis.

John B. Steele, M. D. Volunteer State, Chattanooga, Tenn.

David F. R. Steuart, M. D. Mutual Benefit, Newark, N. J.

Edgar M. Stevenson, M. D. State Farm, Bloomington, Ill.

Lester Q. Stewart, M. D. Aetna, Hartford, Conn.

Hector M. Stevenson, M. D. Aetna, Hartford, Conn.

I. Read Stidger, M. D. Prudential, Newark, N. J.

Frank M. Stites, M. D. Kentucky Home Mutual, Louisville, Ky.

Samuel J. Streight, M. D. Canada Life, Toronto, Canada

Nicholas A. Sullo, M. D. Equitable Life Assurance, New York City.

Bion C. Syverson, M. D. Equitable Life Assurance, New York City

Louis J. Tedesco, M. D. New York Life, New York City

Gamber F. Tegtmeyer, M. D. Northwestern Mutual, Milwaukee, Wis.

Edward R. Thompson, M. D. Texas Prudential, Galveston, Texas

Hugh G. Thompson, M. D. George Washington, Charleston, W. Va.

K. Jefferson Thomson, M. D. Metropolitan, New York City

Walter E. Thornton, M. D. Lincoln National, Ft. Wayne, Ind.

Albert R. Tormey, M. D. National Guardian, Madison, Wis.

Grafton D. Townshend, M. D. Standard Life Association, Lawrence, Kansas

Joseph Travenick, Jr., M. D. Occidental, Los Angeles, Calif.

Wallace Troup, M. D. Metropolitan, New York City

Francis D. Truax, M. D.	Crown, Toronto, Canada
Maurice Turcotte, M. D.	Industrial, Quebec, Canada
Harry E. Ungerleider, M. D.	Equitable Life Assurance, New York City
Bruce W. Vale, M. D.	Excelsior, Toronto, Canada
Euen Van Kleeck, M. D.	Travelers, Hartford, Conn.
Alexander E. Venables, M. D.	Minnesota Mutual, St. Paul, Minn.
Frederick H. Vinup, M. D.	Monumental, Baltimore, Md.
Reynold C. Voss, M. D.	Pan-American, New Orleans, La.
Murray Wagman, M. D.	Mutual Benefit, Newark, N. J.
Frederick A. Waldron, M. D.	Mutual, New York City
George H. Walker, M. D.	Lincoln Liberty, Lincoln, Neb.
Gordon K. Wallace, M. D.	Great American Reserve, Dallas, Tex.
Dick P. Wall, M. D.	American National, Galveston, Texas
Kenneth E. Ward, M. D.	Connecticut General, Hartford, Conn.
R. Vance Ward, M. D.	Montreal Life, Montreal, Canada
Robert L. Weaver, M. D.	Penn Mutual, Philadelphia, Pa.
Jefferson Weed, M. D.	Mutual Benefit, Newark, N. J.
John H. Weidner, M. D.	Prudential, Newark, N. J.
David E. W. Wenstrand, M. D.	Northwestern Mutual, Milwaukee, Wis.
Stephen S. Werth, M. D.	Globe, Chicago, Ill.

Otis B. Wight, M. D.	Standard, Portland, Ore.
Howard E. Wiley, M. D.	Southwestern, Dallas, Texas
John A. Wilhelm, M. D.	Gulf, Jacksonville, Fla.
Alfred A. Willander, M. D.	Mutual Trust, Chicago, Ill.
Earl B. Williams, M. D.	Wisconsin National, Oshkosh, Wis.
Ennion S. Williams, M. D.	Life Insurance Co. of Virginia, Richmond, Va.
Richard L. Willis, M. D.	Mutual, New York City
A. Allison Wills, Jr., M. D.	Travelers, Hartford, Conn.
Archibald C. Wilson, M. D.	Connecticut General, Hartford, Conn.
C. L. Wilson, M. D.	Empire State Mutual, Jamestown, N. Y.
Edmund W. Wilson, M. D.	Metropolitan, New York City
McLeod C. Wilson, M. D.	Travelers, Hartford, Conn.
Don J. Wolfram, M. D.	Jefferson National, Indianapolis, Ind.
George E. Woodford, M. D.	Home, New York City
Lauritz S. Ylvisaker, M. D.	Fidelity Mutual, Philadelphia, Pa.
Donald E. Yochem, M. D.	Farm Bureau, Columbus, Ohio
Arthur W. Young, M. D.	Sun, Montreal, Canada
George G. Young, M. D.	Central, Des Moines, Iowa
Russell W. Zinkann, M. D.	Mutual, Waterloo, Canada

HONORARY MEMBERS

Francis R. Dieuaide, M. D.	New York City
Arthur Hunter	New York City
Edward E. Rhodes	Newark, N. J.

EMERITUS MEMBERS

Edwin H. Allen, M. D.	Boston, Mass.
Thomas D. Archibald, M. D.	Toronto, Canada
William B. Bartlett, M. D.	Boston, Mass.
Chester T. Brown, M. D.	Newark, N. J.
Robert M. Daley, M. D.	New York City
O. M. Eakins, M. D.	Pittsburgh, Pa.
Frank L. Grosvenor, M. D.	Hartford, Conn.
George A. Harlow, M. D.	Milwaukee, Wis.
Byam Hollings, M. D.	Boston, Mass.
Walter A. Jaquith, M. D.	Columbus, Ohio
George E. Kanouse, M. D.	Newark, N. J.
Francis H. McCrudden, M. D.	Boston, Mass.
William Muhlberg, M. D.	Cincinnati, Ohio
Herbert Old, M. D.	Philadelphia, Pa.
George P. Paul, M. D.	Hartford, Conn.
Charles B. Piper, M. D.	Hartford, Conn.
H. Crawford Scadding, M. D.	Toronto, Canada
Samuel B. Scholz, M. D.	Philadelphia, Pa.
Henry H. Schroeder, M. D.	New York City
William R. Ward, M. D.	Newark, N. J.
Fred L. Wells, M. D.	Des Moines, Iowa
Chester F. S. Whitney, M. D.	New York City

COMPANIES AND THEIR REPRESENTATIVES

Acacia Mutual Life Insurance Co., Washington, D. C.	J. R. B. Hutchinson, M. D. J. B. Nichols, M. D.
Aetna Life Insurance Co., Hartford, Conn.	K. F. Brandon, M. D. J. C. Clifford, M. D. J. G. Irving, M. D. H. M. Stevenson, M. D. L. Q. Stewart, M. D.
Alliance Life Insurance Co., Chicago, Ill.	J. E. Hunsinger, M. D.
Alliance Nationale, Montreal, Can.	Bernard Baillargeon, M. D.
All American Assurance Co., Lafayette, La.	Paul Kurzweg, Jr., M. D.
All States Life Insurance Co., Montgomery, Ala.	Bernard Mount, M. D.
American General Life Insurance Co., Houston, Texas	Ghent Graves, M. D. M. L. Graves, M. D.
American Life Insurance Co., Birmingham, Ala.	Cabot Lull, M. D.
American Mutual Life Insurance Co., Des Moines, Iowa.	E. B. Mountain, M. D.
American National Insurance Co., Galveston, Texas.	D. P. Wall, M. D.
American Reserve Life Insurance Co., Omaha, Neb.	R. A. Moser, M. D.

American United Life Insurance Co., Indianapolis, Ind.	Albert Seaton, M. D.
Atlantic Life Insurance Co., Richmond, Va.	Cullen Pitt, M. D.
Baltimore Life Insurance Co., Baltimore, Md.	N. B. Cole, M. D.
Bankers Life Company, Des Moines, Iowa.	A. E. Johann, M. D. George McCreight, M. D. R. E. King, M. D.
Bankers National Life Ins. Co., Montclair, N. J.	B. T. D. Schwarz, M. D.
Bankers Life Insurance Co. of Nebraska, Lincoln, Neb.	H. E. Flansburg, M. D.
Berkshire Life Insurance Co., Pittsfield, Mass.	F. R. Congdon, M. D. Frank Harnden, M. D.
Boston Mutual Life Insurance Co., Boston, Mass.	L. B. Ellis, M. D.
Business Men's Assurance Co. of America, Kansas City, Mo.	C. B. Ahlefeld, M. D.
Canada Life Assurance Co., Toronto, Canada.	A. E. Parks, M. D. S. J. Streight, M. D.
Capitol Life Insurance Co. of Colorado, Denver, Colo.	J. M. Foster, M. D.
Carolina Life Insurance Co., Columbia, S. C.	C. G. Spivey, M. D.
Central Life Assurance Society, Des Moines, Iowa.	M. I. Olsen, M. D. G. G. Young, M. D.
Colonial Life Insurance Co., Jersey City, N. J.	L. A. Pyle, M. D.

Columbian Mutual Life Insurance Co., Memphis, Tenn.	A. R. McMahan, M. D.
Columbian National Life Ins. Co., Boston, Mass.	C. H. Kelley, M. D. F. L. Springer, M. D.
Columbus Mutual Life Ins. Co., Columbus, Ohio.	F. M. Green, M. D.
Commonwealth Life Insurance Co., Louisville, Ky.	A. S. Irving, M. D.
Confederation Life Association, Toronto, Canada.	E. M. Henderson, M. D. F. W. Rolph, M. D.
Connecticut General Life Ins. Co., Hartford, Conn.	N. J. Barker, M. D. C. C. Beach, Jr., M. D. E. A. Keenleyside, M. D. A. J. Robinson, M. D. K. E. Ward, M. D. A. C. Wilson, M. D.
Connecticut Mutual Life Ins. Co., Hartford, Conn.	T. M. Ebers, M. D. H. F. Laramore, M. D. R. E. Nicholson, M. D. H. B. Rollins, M. D. R. C. Secor, M. D.
Conservative Life Ins. Co., Wheeling, W. Va.	E. E. Clovis, M. D.
Constitution Life Company of America, Los Angeles, Calif.	W. E. Branch, M. D.
Continental Amer. Life Ins. Co., Wilmington, Del.	W. M. Genthner, M. D.
Continental Assurance Co., Chicago, Ill.	H. W. Dingman, M. D. C. L. Reeder, M. D.
Continental Life Insurance Co., Toronto, Canada.	S. J. N. Magwood, M. D.
Country Life Insurance Co., Chicago, Ill.	J. E. Boland, M. D.

Crown Life Insurance Co., Toronto, Canada.	H. D. Delamere, M. D. F. D. Truax, M. D.
Dominion Life Assurance Co., Waterloo, Canada	A. J. McGanity, M. D.
Eastern Life Insurance Co., New York, N. Y.	Isaac Sossnitz, M. D.
Empire Life Insurance Co., Kingston, Canada	J. S. Delahaye, M. D.
Empire State Mutual Life Insurance Co., Jamestown, N. Y.	C. L. Wilson, M. D.
Equitable Life Assurance Society, New York City	E. W. Beckwith, M. D. Leslie Brown, M. D. V. J. Fingar, M. D. George Goodkin, M. D. R. S. Gubner, M. D. L. G. LaPointe, M. D. W. J. McNamara, M. D. W. M. Reynolds, M. D. E. W. Scott, M. D. W. A. Smith, M. D. N. A. Sullo, M. D. B. C. Syverson, M. D. H. E. Ungerleider, M. D.
Equitable Life Insurance Co., Washington, D. C.	F. M. McChesney, M. D.
Equitable Life Ins. Co. of Canada, Waterloo, Canada	W. L. Hilliard, M. D.
Equitable Life Insurance Co. of Iowa, Des Moines, Iowa.	W. O. Purdy, M. D. R. R. Simmons, M. D.
Excelsior Life Insurance Co., Toronto, Canada.	W. E. Ferguson, M. D. B. W. Vale, M. D.
Farm Bureau Life Ins. Co., Columbus, Ohio.	D. E. Yochem, M. D.

Farmers & Bankers Life Insurance Co., Wichita, Kan.	J. L. Evans, M. D.
Fidelity Life Assurance Co., Regina, Canada	F. D. Munroe, M. D.
Fidelity Mutual Life Ins. Co., Philadelphia, Pa.	J. T. Sheridan, M. D. L. S. Ylvisaker, M. D.
Fidelity Union Life Insurance Co., Dallas, Texas	J. T. Downs, Jr., M. D.
Franklin Life Ins. Co., Springfield, Ill.	Frederick Fink, M. D.
General American Life Ins. Co., St. Louis, Mo.	J. H. Ready, M. D.
George Washington Life Insurance Co., Charleston, W. Va.	H. G. Thompson, M. D.
Girard Life Insurance Co., Philadelphia, Pa.	J. P. Chapman, M. D.
Globe Life Insurance Co., Chicago, Ill.	S. S. Werth, M. D.
Great American Reserve Insurance Co., Dallas, Tex.	G. K. Wallace, M. D.
Great National Life Insurance Co., Dallas, Texas	P. M. Rattan, M. D.
Great Southern Life Ins. Co., Houston, Texas	N. P. Doak, M. D.
Great-West Life Assur. Co., Winnipeg, Canada.	B. H. Olson, M. D. W. F. H. O'Neill, M. D. F. H. Smith, M. D.
Guarantee Mutual Life Insurance Co., Omaha, Neb.	J. P. Donelan, M. D.
Gulf Life Insurance Co., Jacksonville, Fla.	J. A. Wilhelm, M. D.

Guaranty Income Life Insurance Co., Baton Rouge, La.	H. G. Riche, M. D.
Guardian Life Insurance Co. of America, New York, N. Y.	M. B. Bender, M. D. D. C. Roberts, M. D.
Home Life Insurance Co., New York, N. Y.	J. H. Humphries, M. D. G. E. Woodford, M. D.
Home Life Ins. Co. of America, Philadelphia, Pa.	H. W. Goos, M. D.
Home State Life Insurance Co., Oklahoma City, Okla.	W. W. Rucks, M. D.
Illinois Bankers Life Assurance Co., Monmouth, Ill.	H. G. Ebersole, M. D.
Imperial Life Assurance Co., Toronto, Canada.	R. W. Mann, M. D. D. L. Selby, M. D.
Independent Order of Foresters, Toronto, Canada	C. B. Parker, M. D.
Industrial Life Insurance Co., Quebec, Canada	Maurice Turcotte, M. D.
Interstate Life and Accident Co., Chattanooga, Tenn.	J. W. Johnson, Jr., M. D.
Jefferson National Life Insurance Co., Indianapolis, Ind.	D. J. Wolfram, M. D.
Jefferson Standard Life Ins. Co., Greensboro, N. C.	W. R. Bishop, M. D. H. F. Starr, M. D.
John Hancock Mutual Life Ins. Co., Boston, Mass.	R. A. Behrman, M. D. D. G. Friend, M. D. David Halbersleben, M. D. B. L. Huntington, M. D. R. C. Larcom, Jr., M. D. H. M. Rees, M. D.

Kansas City Life Ins. Co., Kansas City, Mo.	G. P. Barnett, M. D. J. E. Bee, M. D.
Kentucky Home Mutual Life Insurance Co., Louisville, Ky.	F. M. Stites, M. D.
Knights of Columbus, New Haven, Conn.	G. J. Lunz, M. D.
La Sauvegarde Insurance Co., Montreal, Canada.	J. E. Desrochers, M. D.
Liberty Life Insurance Co., Greenville, S. C.	W. S. Fewell, M. D.
Liberty National Life Ins. Co., Birmingham, Ala.	J. A. Livingston, M. D.
Life and Casualty Insurance Co., Nashville, Tenn.	J. J. Post, M. D.
Life Insurance Co. of Georgia, Atlanta, Ga.	O. E. Hanes, M. D.
Life Insurance Co. of Virginia, Richmond, Va.	G. M. Harwood, M. D. E. S. Williams, M. D.
Lincoln Liberty Life Ins. Co., Lincoln, Neb.	G. H. Walker, M. D.
Lincoln National Life Ins. Co., Fort Wayne, Ind.	G. M. Graham, M. D. H. C. McAlister, M. D. W. H. Scoins, M. D. W. E. Thornton, M. D.
London Life Insurance Co., London, Canada.	J. T. Bowman, M. D. A. S. Graham, M. D.
Loyal Protective Life Insurance Co., Boston, Mass.	H. W. Hudson, M. D.
Maccabees (The), Detroit, Mich.	H. R. John, M. D.
Manhattan Life Insurance Co., New York, N. Y.	G. H. Barber, M. D. Eugene V. Higgins, M. D.

Manufacturers Life Ins. Co., Toronto, Canada.	T. C. Dunlop, M. D. H. M. Gray, M. D. R. C. Montgomery, M. D.
Maritime Life Assurance Co., Halifax, Canada	J. G. MacDougall, M. D.
Maryland Life Insurance Co., Baltimore, Md.	G. C. Lockard, M. D.
Massachusetts Mutual Life Insurance Co., Springfield, Mass.	H. B. Brown, M. D. L. D. Chapin, M. D. L. E. Haentzschel, M. D. Gordon Ross, M. D. T. S. Sexton, M. D. Morton Snow, M. D.
Metropolitan Life Insurance Co., New York City	C. C. Berwick, M. D. N. R. Blatherwick, M. D. E. C. Bonnett, M. D. A. W. Bromer, M. D. E. T. Dewey, M. D. J. A. Evans, M. D. R. K. Farnham, M. D. H. H. Fellows, M. D. R. W. Finegan, M. D. J. T. Geiger, M. D. J. C. Horan, M. D. A. O. Jimenis, M. D. H. B. Kidd, M. D. A. J. Lanza, M. D. G. S. Pesquera, M. D. G. P. Robb, M. D. K. J. Thomson, M. D. Wallace Troup, M. D. E. W. Wilson, M. D.
Michigan Life Insurance Co., Detroit, Mich.	R. W. Scott, M. D.
Midland Mutual Life Insurance Co., Columbus, Ohio	P. H. Charlton, M. D.
Midland National Life Insurance Co., Watertown, S. D.	O. S. Randall, M. D.
Midwest Life Insurance Co., Lincoln, Neb.	E. W. Rowe, M. D.

Minnesota Mutual Life Insurance Co., St. Paul, Minn.	T. H. Dickson, M. D. A. E. Venables, M. D.
Missouri Insurance Co., St. Louis, Mo.	C. D. Magee, M. D.
Modern Woodmen of America, Rock Island, Ill.	E. A. Anderson, M. D.
Monarch Life Assur. Co., Winnipeg, Canada	F. A. L. Mathewson, M. D
Monarch Life Insurance Co., Springfield, Mass.	J. M. Gilchrist, M. D. L. E. Hathaway, Jr., M. D.
Montreal Life Insurance Co., Montreal, Canada	R. V. Ward, M. D.
Monumental Life Insurance Co., Baltimore, Md.	F. H. Vinup, M. D.
Mutual Benefit Life Insurance Co., Newark, N. J.	J. R. Beard, M. D. E. C. Hillman, Jr., M. D. W. A. Reiter, M. D. D. F. Steuart, M. D. Murray Wagman, M. D. Jefferson Weed, M. D.
Mutual Life Assur. Co. of Canada, Waterloo, Canada	J. M. Livingston, M. D. R. W. Zinkann, M. D.
Mutual Life Ins. Co. of New York, New York City	H. A. Bancel, M. D. F. V. Costello, M. D. J. R. Gudger, M. D. J. F. Moore, M. D. S. A. Narins, M. D. E. J. Quinn, M. D. F. A. Waldron, M. D. R. L. Willis, M. D.
Mutual Savings Life Insurance Co., St. Louis, Mo.	A. C. Henske, M. D.
Mutual Trust Life Insurance Co., Chicago, Ill.	A. A. Willander, M. D.

National Equity Life Insurance Co., Little Rock, Ark.	W. R. Richardson, M. D.
National Fidelity Life Insurance Co., Kansas City, Mo.	J. V. Bell, M. D.
National Guardian Life Insurance Co., Madison, Wis.	A. R. Tormey, M. D.
National Life & Accident Ins. Co., Nashville, Tenn.	B. F. Byrd, M. D. G. E. Fort, M. D. L. C. Miller, M. D.
National Life Assurance Co. of Canada, Toronto, Canada	E. C. Noble, M. D.
National Life Co., Des Moines, Iowa	L. K. Meredith, M. D.
National Life Insurance Co., Montpelier, Vt.	G. E. Allen, M. D. H. L. Colombo, M. D. A. J. Oberlander, M. D.
National Old Line Insurance Co., Little Rock, Ark.	R. E. McLochlin, M. D.
New England Mutual Life Ins. Co., Boston, Mass.	F. R. Brown, M. D. M. H. Clifford, M. D. H. M. Frost, M. D. O. C. Hendrix, M. D.
New York Life Insurance Co., New York City	D. R. Auten, M. D. M. F. Bell, M. D. William Bolt, M. D. E. J. Campbell, M. D. G. D. Dorman, M. D. A. H. Faber, M. D. E. M. Freeland, M. D. E. E. Getman, M. D. H. L. Hauge, M. D. W. D. Heaton, M. D. T. B. Hoxie, M. D. J. J. Hutchinson, M. D. I. C. Lawler, M. D. R. W. Pratt, M. D. L. J. Tedesco, M. D.

North American Life Assur. Co., Toronto, Canada	J. G. Falconer, M. D. Eugene Montgomery, M. D.
North American Reassurance Co., New York City	J. T. Montgomery, M. D.
Northern Life Assurance Co. of Canada, London, Canada	J. H. Geddes, M. D.
Northwestern Mutual Life Ins. Co., Milwaukee, Wis.	R. W. Benton, M. D. J. A. End, M. D. R. T. Gilchrist, M. D. W. G. Hyde, M. D. D. F. Rikkers, M. D. G. F. Tegtmeyer, M. D. D. E. W. Wenstrand, M. D.
Northwestern National Life Ins. Co., Minneapolis, Minn.	K. W. Anderson, M. D.
Occidental Life Insurance Co., Raleigh, N. C.	V. S. Caviness, M. D.
Occidental Life Ins. Co. of California, Los Angeles, Calif.	E. F. Sheldon, M. D. Joseph Travenick, Jr., M. D.
Ohio National Life Ins. Co., Cincinnati, Ohio	H. H. Shook, M. D.
Ohio State Life Insurance Co., Columbus, Ohio	T. F. Ross, M. D.
Old Line Life Insurance Co. of America, Milwaukee. Wis.	H. M. Hawkins, M. D.
Pacific Mutual Life Ins. Co., Los Angeles, Calif.	L. H. Lee, M. D.
Pan-American Life Ins. Co., New Orleans, La.	Marion Souchon, M. D. R. C. Voss, M. D.

Paul Revere Life Ins. Co., Worcester, Mass.	J. W. Abbott, M. D. Earl J. Kuenster, M. D. H. R. Leffingwell, M. D.
Peninsular Life Insurance Co., Jacksonville, Fla.	E. B. Milam, M. D.
Penn Mutual Life Ins. Co., Philadelphia, Pa.	E. S. Dillon, M. D. J. T. Eads, M. D. D. W. Hoare, M. D. C. F. Nichols, M. D. D. M. Shewbrooks, M. D. R. L. Weaver, M. D.
Peoples Life Ins. Co., Frankfort, Ind.	C. A. Robison, M. D.
Peoples Life Insurance Co., Washington, D. C.	J. R. Biggs, M. D.
Philadelphia Life Ins. Co., Philadelphia, Pa.	T. M. Armstrong, M. D.
Phoenix Mutual Life Ins. Co., Hartford, Conn.	H. B. Campbell, M. D. R. A. Goodell, M. D. Llewellyn Hall, M. D. N. R. Kelley, M. D. R. L. Rowley, M. D.
Pilot Life Insurance Co., Greensboro, N. C.	J. L. Cook, M. D.
Pioneer Mutual Life Insurance Co., Fargo, N. D.	F. O. Gronvold, M. D.
Policyholder's National Life Ins. Co., Sioux Falls, S. D.	Rezin Reagan, M. D.
Postal Life Insurance Co., New York, N. Y.	L. B. Dunn, M. D.
Praetorians (The), Dallas, Texas	E. P. Leeper, M. D.

Preferred Life Assurance Society, Montgomery, Ala.	B. C. Bird, M. D.
Protected Home Circle, Sharon, Pa.	W. G. McLaughry, M. D.
Protective Life Insurance Co., Birmingham, Ala.	E. G. Givhan, Jr., M. D.
Provident Life and Accident Ins. Co., Chattanooga, Tenn.	C. R. Henry, M. D.
Provident Mutual Life Ins. Co., Philadelphia, Pa.	E. J. Dewees, M. D. P. H. Langner, Jr., M. D. L. L. McLellan, M. D. D. S. Pepper, M. D.
Prudential Assur. Co., Ltd., Montreal, Canada	C. A. Peters, M. D.
Prudential Insurance Co., Newark, N. J.	R. F. Buchan, M. D. P. E. Carlisle, M. D. E. G. Dewis, M. D. A. H. Domm, M. D. F. I. Ganot, M. D. W. C. Hausheer, M. D. E. G. Howe, M. D. C. E. Kiessling, M. D. H. B. Kirkland, M. D. F. J. McGurl, M. D. R. A. Nelson, M. D. W. C. Page, M. D. P. V. Reinartz, M. D. K. F. Schaefer, M. D. I. R. Stidger, M. D. J. H. Weidner, M. D.
Puritan Life Insurance Co., Providence, R. I.	E. D. Chesebro, M. D.
Pyramid Life Insurance Co., Little Rock, Ark.	J. H. Sanderlin, M. D.
Reliance Insurance Co. of Pittsburgh, Pittsburgh, Pa.	H. A. Cochran, Jr., M. D. I. Kenneth Gardner, M. D. J. L. Humphreys, M. D.

Republic National Life Ins. Co., Dallas, Texas	D. G. Kilgore, M. D.
Reserve Loan Life Ins. Co., Dallas, Texas	D. W. Carter, Jr., M. D.
Rockford Life Insurance Co., Rockford, Ill.	P. A. Anderson, M. D.
Royal Insurance Co., Ltd., Montreal, Canada	G. W. Halpenny, M. D.
Scranton Life Insurance Co., Scranton, Pa.	G. G. Lindsay, M. D.
Security Life and Accident Co., Denver, Colo.	D. S. Baughman, M. D. R. C. Scannell, M. D.
Security Life & Trust Co., Winston-Salem, N. C.	J. P. Davis, M. D. S. W. Hurdle, M. D.
Security Mutual Life Ins. Co., Binghamton, N. Y.	W. B. Aten, M. D.
Shenandoah Life Insurance Co., Inc., Roanoke, Va.	D. S. Garner, M. D.
Southern Life Insurance Co. of Georgia, Atlanta, Ga.	D. Y. Sage, M. D.
Southland Life Insurance Co., Dallas, Texas	Hall Shannon, M. D.
Southwestern Life Ins. Co., Dallas, Texas	C. F. Brown, M. D. H. E. Wiley, M. D.
Standard Insurance Company, Portland, Oregon	O. B. Wight, M. D.
Standard Life Association Lawrence, Kansas	G. D. Townshend, M. D.
Standard Life Assur. Co., Montreal, Canada	W. W. Eakin, M. D.

State Farm Life Insurance Co., Bloomington, Ill.	J. K. P. Hawks, M. D. E. M. Stevenson, M. D.
State Life Insurance Co., Indianapolis, Ind.	F. T. Hallam, M. D. C. B. McCulloch, M. D.
State Mutual Life Assur. Co., Worcester, Mass.	H. H. Amiral, M. D. F. P. Bicknell, M. D.
State Reserve Life Insurance Co., Fort Worth, Texas	Samuel Jagoda, M. D.
Sun Life Assurance Company of Canada, Montreal, Canada	C. C. Birchard, M. D. J. K. Gordon, M. D. A. W. Young, M. D.
Sun Life Insurance Co. of America, Baltimore, Md.	George McLean, M. D.
T. Eaton Life Assurance Co., Toronto, Canada	C. V. Mulligan, M. D.
Texas Life Insurance Co., Waco, Texas	I. E. Colgin, M. D.
Texas Prudential Insurance Co., Galveston, Texas	E. R. Thompson, M. D.
Toronto Mutual Life Insurance Co., Toronto, Canada	W. W. Dow, M. D.
Travelers Insurance Company, Hartford, Conn.	J. T. Cabaniss, M. D. R. M. Filson, M. D. L. C. Grau, M. D. A. L. Larson, M. D. Euen van Kleeck, M. D. A. A. Wills, M. D. M. C. Wilson, M. D.
Union Central Life Insurance Co., Cincinnati, Ohio	William Brueggemann, M. D. Edward Kuck, M. D. Charles Maertz, M. D.
Union Labor Life Insurance Co., New York, N. Y.	W. L. O'Connell, M. D.
Union Life Insurance Co., Little Rock, Ark.	J. H. Hayes, M. D.
Union Mutual Life Insurance Co., Portland, Maine	H. E. Christensen, M. D.

United Benefit Life Insurance Co., Omaha, Neb.	N. L. Criss, M. D.
United Fidelity Life Insurance Co., Dallas, Texas	H. K. Crutcher, M. D.
United Life and Accident Ins. Co., Concord, N. H.	H. H. Amsden, M. D. P. M. L. Forsberg, M. D. R. J. Graves, M. D.
United States Life Ins. Co., New York City	J. A. Avrack, M. D. M. J. O'Brien, M. D.
Volunteer State Life Ins. Co., Chattanooga, Tenn.	J. B. Steele, M. D.
Washington National Insurance Company, Evanston, Ill.	I. E. Hoffman, M. D.
West Coast Life Ins. Co., San Francisco, Calif.	I. C. Heron, M. D.
Western Life Assurance Company, Hamilton, Canada	G. E. Greenway, M. D.
Western Life Insurance Company, Helena, Mont.	T. L. Hawkins, M. D. E. H. Lindstrom, M. D.
Western Reserve Life Insurance Company, Austin, Texas	C. P. Hardwicke, M. D.
Western and Southern Life Ins. Co., Cincinnati, Ohio	C. M. Barrett, M. D. E. R. Bush, M. D.
Western States Life Insurance Company, Fargo, N. D.	T. H. Lewis, M. D.
Wisconsin Life Insurance Company, Madison, Wis.	G. G. Stebbins, M. D.
Wisconsin National Life Insurance Company, Oshkosh, Wis.	E. B. Williams, M. D.
Woodmen of the World Life Insurance Society, Omaha, Neb.	A. D. Cloyd, M. D. H. B. Kennedy, M. D.

AUTHOR INDEX

Vol. XXXII—1948

Note—Titles of Original Papers in Plain Type;
of Papers Discussed, in *Italics.*

SUBJECT INDEX

Vol. XXXII—1948

Note—The Names of Authors are Printed in Plain Type; of Discussers, in *Italics.*